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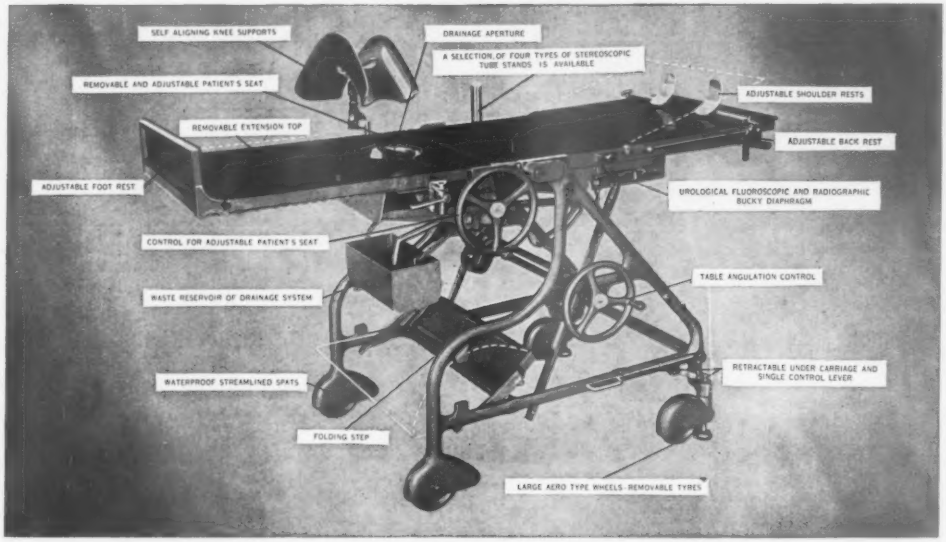
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Hill (*Medical Journal of Australia*, 9.5.36, page 656) reported a small series treated with Antuitrin-S and states "that all patients should be given the chance of this treatment before operation".

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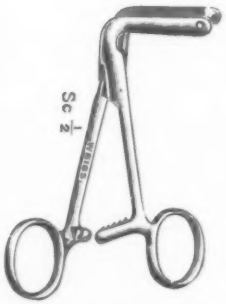

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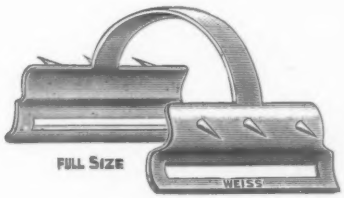

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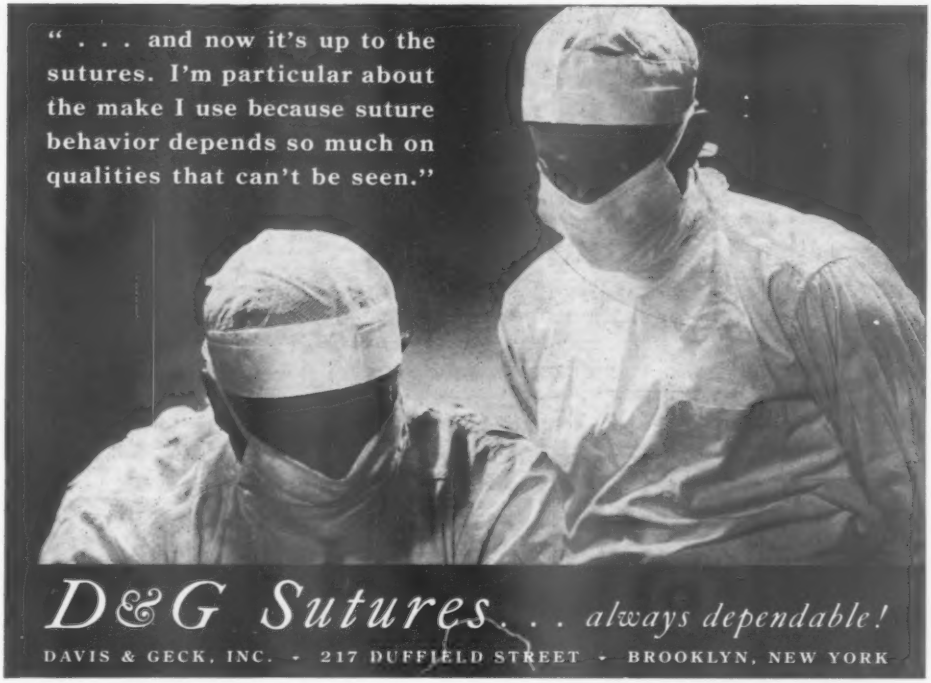
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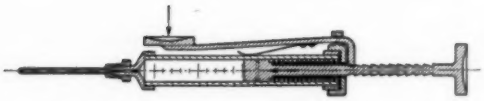

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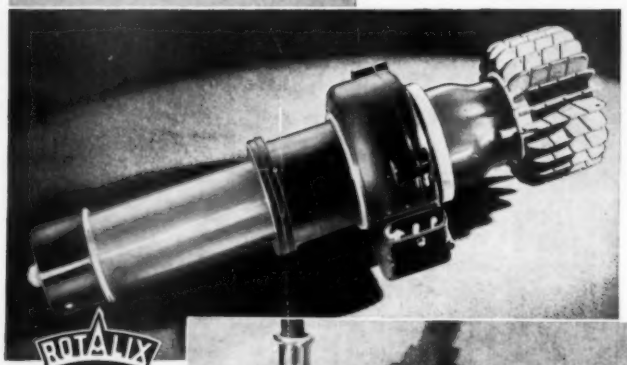

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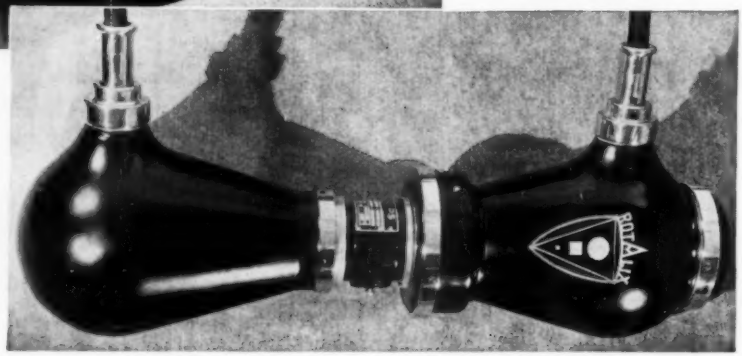

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THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY

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HYDATID CYSTS INVOLVING THE DOME OF THE LIVER.[1]

By SIR LOUIS BARNETT,

Emeritus Professor of Surgery, University of Otago, New Zealand.

ECHINOCOCCOSIS, or hydatid infection in man and animals, has been and continues to be a subject of such fascinating interest to so many zoologists, clinicians, pathologists, radiologists, hygienists and laboratory research workers in so many parts of the world, that the literature devoted to it, especially the foreign literature, has reached colossal proportions. The world-famous Dévé, of Rouen, for example, has published nearly 300 articles, large and small, which deal in masterly fashion with practically every aspect of this remarkable parasitic malady.[1][2]

There are hundreds of other foreign writers and a few British, including notably Professor H. R. Dew, of Sydney, whose modern and valuable work is well known to us all.[3] Much of this spate of literature is, to my mind, needlessly meticulous and its reading is laborious. I venture to think that the study of hydatid disease from the clinical point of view at any rate could be greatly simplified if more stress was laid on broad principles and if less attention was paid to the details which the reader ought to be able to fill in for himself.

To illustrate this idea I have selected for your attention the cysts located in the region of the dome of the liver, because they in particular are liable to complications which should be largely self-explanatory, but which bewilder the clinical observers and operators who do not keep in mind (*a*) the biology of the hydatid parasite, (*b*) its anatomical environment, (*c*) the natural evolution of the cyst and the natural reactions of the host.

BIOLOGY.

Just a few words about the biology of the parasite, as bearing on the location of cysts. The eggs of the *Tænia echinococcus* hatch out in the upper part of the intestine of the human being, who is unfortunate

[1] Read at the annual meeting of the Royal Australasian College of Surgeons, Auckland, January, 1937.

enough to swallow them, into microscopic embryos, possessing three pairs of boring spikes and an urge to use them. They delve into the mucous membrane until they reach a blood vessel, usually a radicle of a portal vein, and there they get a free ride in the blood stream to the liver capillaries. There, most of them stick, and if they have vitality enough to overcome the defensive reactions of their host, these little "gate-crashers" settle down and grow into cysts. About 75% of them are thus filtered out in the liver. The rest are carried on in the blood stream to the next stop, the lungs, where about 10% similarly stick in the pulmonary capillaries, and the small balance, the long-distance travellers, pass on into the systemic capillaries and give rise to cysts in various other parts of the body (Dévé[4]).

This is the anatomical, but not whole, explanation of the distribution of primary hydatid cysts. Biological affinity also plays an important part. The liver is not the fashionable residential quarter in all animals. In the squirrel, for instance, hydatid cysts are nearly always found in the lung, and, in cattle, lung cysts are much commoner than liver cysts.

Two other points must be borne in mind. One is that children are much more susceptible to worm infection than adults, and the other is that only a very small proportion of the hydatid ova that are swallowed actually develop into cysts. Most of them die in infancy. If hydatid cysts are to grow, they must possess strong vitality, and they must find a host that does not resent their intrusion. He or she must be susceptible to infection.

My impression is that only a proportion, perhaps a small proportion, of the human beings who swallow hydatid ova subsequently suffer from hydatid cysts. The majority have an inherited or acquired immunity.

The problem of natural and artificial immunity to hydatid infection offers a fertile field for research. Turner, Dennis and Berberian,[5] of the American University of Beirut, Syria, have in recent years been working on this subject, and report that with a stabilized antigen they have produced a small but definite degree of immunity in dogs and in sheep. Dévé, of Rouen, had previously done some work in this direction, but was disappointed with his results. Our own work at the New Zealand Medical School has shown us that, without any artificial aid, about 66% of all dogs are naturally immune.

ANATOMICAL ENVIRONMENT.

Now, the liver is a large organ and one may reasonably say that the region of the dome occupies about one-quarter of its bulk, and consequently about one-quarter of the liver cysts occur in this special location. The region of the dome is an ideal home for the hydatid cyst. This so-called silent zone of the liver is so well sheltered under the lower thoracic walls and is so hidden from view, that the cyst can, and commonly does, grow larger and larger in the course of years until it reaches perhaps the size of an ostrich egg before it is observed. It is usually so quiet and well-behaved that no obvious manifestations of

ill-health are produced until the patient, who probably was infected in childhood, reaches adult or middle age, when complications are likely to cause trouble.

This latency in clinical manifestation leaves the cyst free from any surgical intervention for ten, twenty or thirty years, and gives unrivalled opportunities for the final development of pathological complications.

Quietly, steadily, relentlessly, with its high intracystic pressure, the cyst extends in centrifugal fashion. It bulges from the liver upwards into the thorax, displacing and sometimes penetrating the diaphragm; and thus it may lead to all sorts of pulmonary involvement. This tendency to upward bulging is probably favoured by the negative pressure exerted by the respiratory mechanism. The cyst makes the ribs and cartilages of the lower thoracic wall bulge outwards, often in very marked degree, and it also pushes the liver downwards into the abdominal cavity, well below its normal level. If, as usual, the right lobe of the liver is involved and its structure and function are damaged, the left lobe becomes enlarged by a process of compensatory hypertrophy. This hypertrophy may be so pronounced as to cause diagnostic errors.

THE NATURAL EVOLUTION OF THE CYST AND THE NATURAL REACTIONS OF THE HOST.

The typical undamaged hydatid cyst consists of a water bladder formed by the parasite membranes, a thin protoplasmic germinal layer called endocyst internally, and a thick elastic laminated hyaline layer called the ectocyst externally, enclosing at high tension the clear watery hydatid fluid. Surrounding this parasitic cyst is a fibro-cellular envelope formed by the tissues of the host. This is called the adventitia or pericyst, and although in close contact, it has no organic connexion with the ectocyst. There is a potential space between them, but in an undamaged cyst there is no actual space, because the high intracystic pressure of the hydatid fluid, which is much higher than blood pressure, keeps the hydatid membranes pressed closely against the adventitia just as the rubber bladder inside a football is kept by air pressure in close contact with the leather cover (see Figure I).

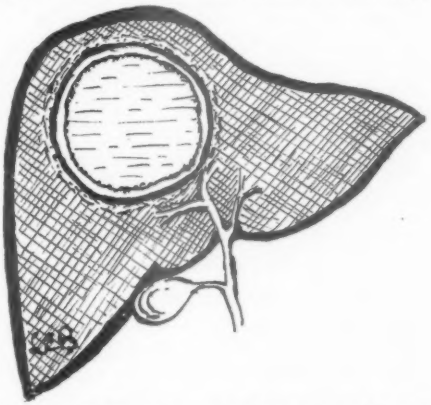

FIGURE I. Juvenile cyst, a few years old only, in the region of the dome of the liver—"*la zone silencieuse*" of Dévé. The contained hydatid fluid is clear and at high tension (considerably above blood pressure), and the hydatid membranes are thereby kept in close contact with the adventitia.

A typical cyst is for many years aseptic, contains clear

fluid at high tension and has no daughter cysts, though brood capsules and scolices may be budded off in vast numbers by the endocyst. This simple univesicular cyst behaves very much like a benign neoplasm, and the only clinical features, if any, likely to be observed are those resulting from the slow but unceasing centrifugal pressure. If by chance an X ray examination is made, the upward bulging of the diaphragm may be seen; and if by chance laboratory tests are made, positive evidence of hydatid infection may be revealed. Ordinarily, then, for many years the patient complains of nothing worth mentioning, and the doctor observes nothing worth mentioning.

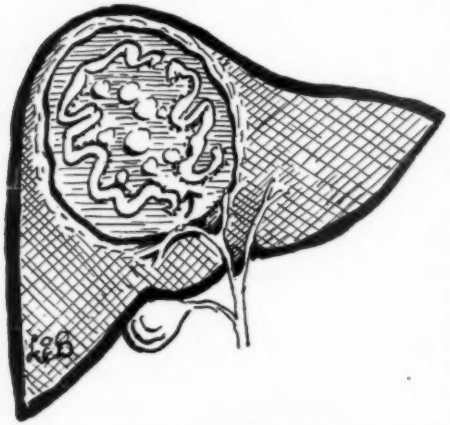

FIGURE II. Cyst after growing for many years, say, ten years or more. Sooner or later, following some injury to the cyst, or maybe resulting from some degeneration due to malnutrition associated with fibrous thickening and calcification in the adventitia, a fissure occurs in the brittle parasitic membranes and they crumple up inside the cyst cavity. The normal osmosis is altered, the intracystic pressure falls, bile leaks in and may introduce infection. The endocyst, the germinal layer of the parasitic membranes, menaced in its vitality, reacts by developing daughters. *"Tout kyste hydatique à contenu multivésiculaire est un kyste ayant souffert"* (Dévé).

But sooner or later something happens that profoundly alters the pathology and the clinical picture, and this something may be either a traumatism or a degenerative process associated with old age of the parasite.

We all know that small leakages or frank rupture of a liver cyst into the peritoneal cavity may result from injury, or muscular strain, and that serious acute or chronic abdominal troubles, such as anaphylactic and toxic shock, secondary peritoneal implantations and so on, may be thereby caused. A rupture of this kind is not very common in the well sheltered cysts located high up in the liver, but it does occur there and unfortunately is sometimes overlooked in the search for a cause of an acute abdominal syndrome.

What is, however, very common in hydatid cysts located deep in the liver, as elsewhere, is a minor degree of traumatism which tears or makes a fissure in the very fragile friable membranes only and not in the tough fibrous adventitia, and which by so doing ushers in a whole series of pathological troubles (see Figure II).

The torn membranes part company from the adventitia, the osmotic control exerted by the ectocyst is lost, intracystic pressure falls, the vitality of the parasite is threatened, and, as a provision for the perpetuation of the species, scolices are transformed into daughter cysts.

The mother membranes degenerate and the simple univesicular cyst becomes converted into a complicated multivesicular cyst. That does not matter very much if the cyst remains aseptic. It may actually die an aseptic death from inanition and be quietly and harmlessly entombed in the thickened fibrous adventitia, which, if calcified, forms then its stony sepulchre (see Figure III).

FIGURE III. X ray picture of an old cyst of the right dome of the liver showing extensive calcification of the adventitia.

These quiet aseptic calcified hydatid cysts are discovered every now and then as surprise *post mortem* findings, having given rise to no particular trouble during life. If, by chance, they are diagnosed during life they are best let alone.

It is a very different story when sepsis is introduced, for sepsis is the chief cause of all the many grave troubles associated with deep-

seated liver cysts, including those of the dome. Sepsis almost invariably comes about through the ingress of infected bile.

As the cyst grows, its steady and relentless pressure causes erosion of the walls of the bile ducts included in the surrounding adventitia. So long as the hydatid membranes are maintained by the high intracystic pressure in close contact with the adventitia, no leakage of bile can take place; but when as a result of injury or degeneration the membranes fall inwards and lose this contact, there is nothing to prevent bile seeping

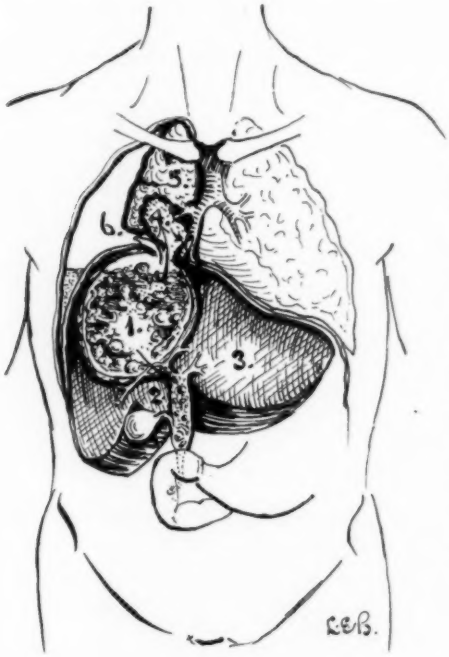

FIGURE IV. Cyst very old, say, twenty years or more. 1. Note the adventitia much thickened and partly necrotic, partly calcified. The contents are living and dead daughters, broken down degenerated membranes, bile and pus. The irregularities in the horizontal surface line caused by the floating daughter cysts and membranes may show on radioscopic view as the sign of the "camalote" (Lagos Garcia and Segores). ("El camalote" is the name given to heaving masses of water weed and débris that in times of flood float down the Rio de la Plata.) 2. A large perforation is shown below, through which cyst contents are evacuated into the main bile ducts, distending them and causing biliary colic, jaundice *et cetera*. 3. The left lobe of the liver shows compensatory hypertrophy. 4. A large perforation is shown above extending through the diaphragm into the right lung and into a bronchus, causing pulmonary abscess, hepato-bronchial fistula, putrid vomica *et cetera*. 5. The right lung is shown partly collapsed. 6. Perforation has also occurred into the right pleural cavity, causing pyopneumothorax.

into the interior of the cyst. Now, bile under normal conditions may be free of bacterial contamination, but when a hydatid cyst is present it is only too likely to harbour septic germs.

An investigation of the bile under these conditions may reveal the presence of a large variety of bacteria, most of them aerobic, such as the *Bacillus coli*, staphylococcus, streptococcus, pneumococcus and the bacillus of Eberth, some of them anaerobic and gas-producing, such as the *Bacillus coli* (facultative), *Staphylococcus parvulus, Streptococcus tenuis, Bacillus nebulosus, Bacillus perfringens, Micrococcus fœtidus et cetera.*

This bacterial infection varies in virulence. In most cases it is low grade, and falls short of causing frank suppuration; but sometimes it is intensely acute and even necrotic in effect. The cyst may become converted into a putrid abscess which may then extend in various directions, penetrate the diaphragm, and, just as one would expect, may cause pleurisy, empyema, pneumonia, pulmonary abscess, gangrene, hepato-bronchial fistula, hepatitis, cholangitis, pylephlebitis, subphrenic suppuration, jaundice, toxæmia, pericarditis, mediastinitis, and so on. All these complications are particularly liable to occur in old-standing cysts of the dome of the liver; and, as I have already explained, cysts in this region, when met with clinically, are usually of very old standing.

The erosion of bile ducts in the thick adventitious capsule is sometimes on the grand scale. Large bile ducts are opened into and large openings are made, so that it becomes not merely a matter of seepage of bile into the cyst cavity, but of evacuation of the contents of the cyst into and down the bile ducts (see Figure IV).

This is really not an uncommon occurrence, and Dévé estimates that it occurs in about 10% of liver cysts and comes second in frequency amongst the complications, rupture into the peritoneal cavity being at the head of the list. Dew places it first. It certainly is very common, and a remarkable point about it is that the cysts which open in this way into large bile ducts are in quite 75% of the cases located deep and high in the liver substance, and not as one might expect in the region of the portal fissure, main bile ducts and gall-bladder.

This high location of the leaking cyst accounts for frequent errors in diagnosis and in treatment; for even after the abdomen has been opened, the existence of the cyst may not be recognized. All too often the jaundice and the biliary colic, associated with the evacuation by way of the bile ducts of hydatid membrane and daughter cysts, are erroneously ascribed to gall-stones, and the true diagnosis is missed.

Much has been written on the opening of hydatid cysts into bile ducts, the complete

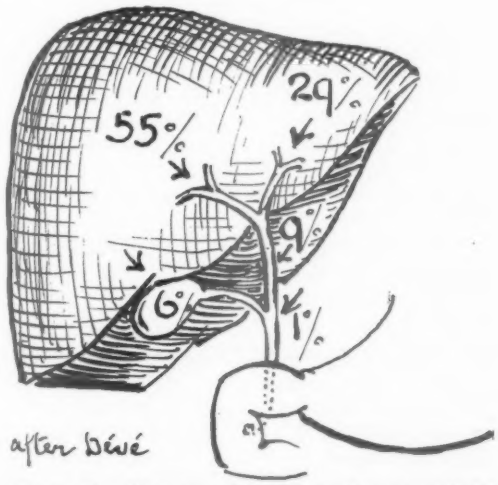

FIGURE V. Diagram with comparative percentages showing the site of the opening of a hydatid cyst of the liver into bile ducts. Note the rarity of opening into the extra-hepatic ducts.

or more commonly incomplete and fractional evacuation by this route, the associated distension of ducts and gall-bladder, the combination with gall-stones, the occurrence of sepsis leading to cholangitis, hepatic suppuration, septicæmia *et cetera*.

Suffice it to say in this paper that cysts of the dome of the liver, for reasons which I hope I have made obvious, are just those in which such complications are particularly likely to be encountered.

The erosion of the walls of blood vessels in the adventitia of an expanding and possibly suppurating cyst takes place in the same fashion as with bile ducts, and in some cases, fortunately rare, a vessel like the *vena cava* or a large branch of the portal or hepatic veins may be opened up with fatal results from hæmorrhage or from hydatid pulmonary embolism. There are records of such cases in the Hydatid Registry. Usually, however, blood vessels in the adventitia are flattened out and fibrosed, and their endothelium-lined lumen is obliterated as a result of the long-standing pressure on their walls. Otherwise serious hæmorrhage would be a much commoner complication than it is. The bile ducts are not obliterated in the same way. In spite of pressure, their lumina remain patent because of their protective lining of epithelium.

DIAGNOSIS.

I could not possibly give adequate consideration to the subject of diagnosis in the time at my disposal, but I do wish to stress the following points:

1. Always keep in mind the possibility of a hydatid cyst being the cause of liver and lower thoracic troubles: cultivate an alert hydatid conscience.
2. The X ray picture will reveal an upward bulging of the diaphragm in the affected region, with comparative lessening of diaphragmatic movement, but these findings may be obscured by associated pleuritic effusion. Artificial pneumothorax or pneumoperitoneum may be useful in cases of special difficulty.
3. Laboratory tests will in most cases give positive evidence of hydatid infection.
4. Where jaundice and biliary colic are present, have the stools examined carefully for the presence of hydatid material. Duodenal tubage may also be helpful.

TREATMENT.

Treatment should, of course, be operation at the earliest favourable opportunity after the diagnosis is made. The sooner a deep-seated liver cyst is evacuated, the better will be the patient's chances of survival and recovery of good health. In old-standing cases there is a heavy mortality; and in those patients who survive, complete recovery is delayed for months and sometimes for years.

Dévé, of Rouen, Dew, of Sydney, Lozano, of Saragossa,[6] and many other authorities state in no uncertain terms that cysts of the dome of

the liver should always be dealt with by the transthoracic route, portions of one or more of the lower ribs as may be necessary being excised, and the pleura and diaphragm being then traversed. They argue that the cysts can be reached more easily and with less risk of hæmorrhage by the thoracic approach and can be drained more efficiently than when the abdominal approach is used.

Most surgeons, I think, follow this counsel; but I venture to doubt its wisdom, as do Lehmann, of Rostock, and some others.[7] My own opinion, based on personal experience and on a study of the Hydatid Registry, is that as a general rule all liver cysts, including those located in the region of the dome, should be dealt with through an abdominal incision which, to begin with, is exploratory and which may be extended, if need be, upwards through one or two of the lower costal cartilages.

The transthoracic should be the exceptional mode of approach, and should be reserved for two types of hydatid cysts located high in the liver: (i) cysts in which thoracic complications, such as penetration of diaphragm, empyema or lung abscess, are already present; (ii) cysts in which an abdominal exploration shows that the cyst is located so high up and so far back that it cannot be approached except by the transthoracic route.

I have formed the impression that most of the cysts that make the diaphragm bulge upwards can be reached by way of the abdomen because, as a rule, they are large cysts and bulge downwards as well as upwards. When their position is revealed by abdominal exploration, the thickness of the intervening liver substance can be estimated, an exploring needle being used if need be, and then a judgement can be made as to the necessity for transthoracic evacuation. Sir Hugh Devine, of Melbourne, tells me that he finds ready access with the help of his special retractor to cysts of the dome by way of the abdominal incision.

Judging by the results of operation, the transthoracic operation on liver cysts is distinctly more dangerous than the abdominal, as you will see from this series of records taken from the Hydatid Registry, which shows that just about twice as many patients died after the transthoracic as after the abdominal technique.

Of the first 1,024 cases recorded in the Hydatid Registry of the Royal Australasian College of Surgeons, 697 were instances of hydatid cysts of the liver.

In 493 of these the ordinary abdominal operation was performed with a mortality of 50. That is to say, just about one in every 10 was fatal.

In 123 the transthoracic operation for cyst of the dome of the liver was performed with a mortality of 27. That is to say, just about one in every five was fatal.

(In 81 of the 697 cases there had been leakage or rupture into the peritoneal cavity prior to operation, so these are not included in the above comparison.)

I admit that this conclusion cannot be accepted without reservation, because most of the cysts approached by thoracic incision were old standing and consequently often septic and complicated, whereas most of those dealt with through the abdominal route were younger and simpler in type. Nevertheless, it does look as if there were definitely more risks in the thoracic than in the abdominal approach.

In the thoracic operation the pleural cavity is nearly always contaminated by the cyst contents, for the efforts to prevent this by preliminary suturing or by gauze packing, when the two-stage technique is employed, are rarely successful. Now, the pleura, as is well known, has much poorer resistance to sepsis than has the peritoneum, and empyema and pyopneumothorax consequently are frequent and, of course, serious complications. The adjacent lung, too, may be involved, and septic lobar or bronchopneumonia set up.

Most of the deaths recorded have resulted from septic pulmonary complications of this kind.

Dévé, from the study of the work and writings of his surgical colleagues, for Dévé himself is a physician, considers that the abdominal evacuation of cysts located high in the liver involves a special risk of hæmorrhage because of the thickness of liver tissue through which the surgeon may have to cut. This risk would be lessened if a cautery knife were used, but it is not at all certain that hæmorrhage is more to be dreaded in the abdominal than in the thoracic cases.

The cases I have tabulated show that the same number (five) of fatalities from hæmorrhage occurred after the 123 transthoracic operations as after the 493 abdominal operations.

Finally, an important point to bear in mind is that in a large proportion of the cases there is more than one liver cyst present, and without an abdominal exploration this contingency might easily be missed.

Time and space do not permit any detailed description of operative technique, and I shall limit myself here to the consideration of just a few practical points.

1. For the abdominal approach which I maintain should be the operation of election, a right paramedial incision usually suffices. This incision extends from the costal margin as far downwards as is adequate for the introduction of the whole hand into the abdominal cavity. The thorough and gentle exploration of the whole abdomen can then be carried out, other cysts and other lesions may be revealed, and a decision made as to the advisability in exceptional cases of switching over from an abdominal to a thoracic operation. In the latter eventuality the operator must use his judgement in individual cases as to whether this thoracic operation should be performed forthwith or deferred until the abdominal wound has healed.

If need be, the paramedial incision can be extended upwards for an inch or two, the costal cartilages being divided close to the sternum.

I have found that this simple extension with upward and outward retraction of the costal margin gives good access to the upper parts of the liver; and, moreover, there is not much likelihood of opening into the pleural cavity, which here is especially protected by a thick layer of extrapleural fatty fascia, as Professor Gowland, of the Department of Anatomy at the New Zealand Medical School, has demonstrated to me.

Costantini,[8] of Algiers, recommends an incision parallel with the costal margin, with, when necessary, an oblique extension upwards along the line of an appropriate intercostal space. Through the abdominal part of this incision the diaphragm can be digitally pressed upwards and outwards, and the pleural cavity thus safeguarded. Ivanissevich,[9] of Montevideo, in an excellently written and well illustrated monograph, describes other methods of dealing with cysts of the dome of the liver by abdominal and thoracic routes.

2. As regards the commonly employed thoracic approach to cysts of the dome, an approach which in my opinion should be exceptional, I wish to stress the following points: (*a*) Local anæsthesia is preferable to general anæsthesia, though there is said to be less risk of anaphylactic shock with the latter. If general anæsthesia is used, intratracheal administration is advisable. (*b*) With local anæsthesia the jockey position as recommended by Lamas, of Buenos Aires, may be helpful. (*c*) The position of the thoracic incision, and the resection of portions of one or two of the lower ribs, must be planned with due regard to the X ray location of the cyst, and should, of course, be as low as possible. (*d*) With the one-stage technique the pleural cavity is likely to be opened. Protective sutures do not hold well in the flimsy pleura, and it is advisable to sew the edges of the cuts in the diaphragm and adventitia to the intercostal musculo-fascial layer. (*e*) The two-stage technique employed with a view to obtaining preliminary obliteration of the pleural cavity before an opening is made into a probably infective liver cyst, seems sound in theory, but in practice, judging from the records I have studied, there does not seem to be any definite advantage over the one-stage procedure.

3. *Formolage,*[10] first recommended by Dévé with a view to lessening the risk of scolex implantation, is of great value in dealing with univesicular cysts, but is practically useless in old cysts packed with daughters and degenerated membranes. Needless to say, formalin is especially contraindicated where a bronchial fistula is present.

4. The evacuation of the contents of the cyst requires patient and painstaking work with scoop and forceps, aided by the use of large-bore suction tube and swabs wrung out in 2% formalin solution.

5. Before concluding that the cyst is really empty, look inside with the aid of retractors, headlight or endoscope, because outlying pockets containing exogenous daughters or hydatid *débris* hidden in their depths are by no means uncommon.

6. Old multivesicular cysts should be drained by the use of a good large suitably placed rubber tube in a dependent position. Drainage by the abdominal route is more likely to be efficient than by the thoracic, when the patient is up and about. Defective drainage is common and sometimes difficult to avoid, and chronic sinuses may persist for months and even years, especially when the adventitia has been extensively calcified. Secondary operations for improving the drainage, removing necrotic and calcified fragments and aiding the obliteration of the cavity are necessary in many cases before final healing is obtained, and in some cases, alas, healing never occurs.

SUMMARY.

A simplified explanation based on broad principles is given of the numerous and sometimes puzzling complications associated with old-standing hydatid cysts of the dome of the liver. Attention is called to the clinical latency of these cysts over a long period, to errors in diagnosis, to the evil influence of bile contamination, and to the pros and cons of the abdominal and thoracic lines of approach in operating.

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ISCHÆMIA.[1]

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ANYONE dealing extensively with the surgery of the limbs in which extensive manipulations or open operations are followed by encasement of the limb in a plaster cast, must have constantly in mind the possibility of the occurrence of ischæmia. A knowledge of the pathology of the condition gives the key to treatment, and fortunately this knowledge is available in recent years.

The two best known examples of ischæmia met with in practice are Volkmann's ischæmia in the forearm and hand, and congenital wry-neck, due to ischæmic contracture of the sterno-mastoid muscle. It is in the search for an explanation of these two conditions that the pathology of ischæmia has been worked out. Volkmann first described the condition in the forearm in 1875, and thought that the cause was the cutting off of arterial blood supply, resulting in a lack of oxygen in the muscles; he compared the condition to *rigor mortis*.

Later, Jepson failed to produce the condition by tightly constricting the hind limbs of dogs; he succeeded, however, in producing typical ischæmic contracture if, in addition to tight constriction of the limb, ligation of the femoral vein was carried out. He followed this up by an observation that the development of ischæmia in the above circumstances could be prevented by making suitable incisions in the deep fascia.

FIGURE I. Sterno-mastoid tumour excised from child aged six weeks, showing fibrous tissue replacing muscle. (After D. Stewart Middleton.)

In cases of congenital torticollis, a large, hard, tender tumour of the sterno-mastoid muscle is noticed at birth. This gradually disappears, and is followed by the development of a scarred, hard, contracted sterno-mastoid muscle (Figure I). In many textbooks an explanation of this phenomenon is still offered by the view that a hæmatoma develops in the muscle, and leads later to scarring and the typical contracture.

[1] The substance of a paper read at a meeting of the Queensland Fellows of the Royal Australasian College of Surgeons on August 14, 1935.

When one contemplates for a moment the extensive hæmatomata that occur in muscles as a result of football injuries, and the rapidity with which they clear up, this explanation becomes untenable. If a hæmatoma in muscle led to the extensive scarring seen in the sterno-mastoid muscle in cases of wry-neck, then most of our first-class athletes would be crippled in one season of sport.

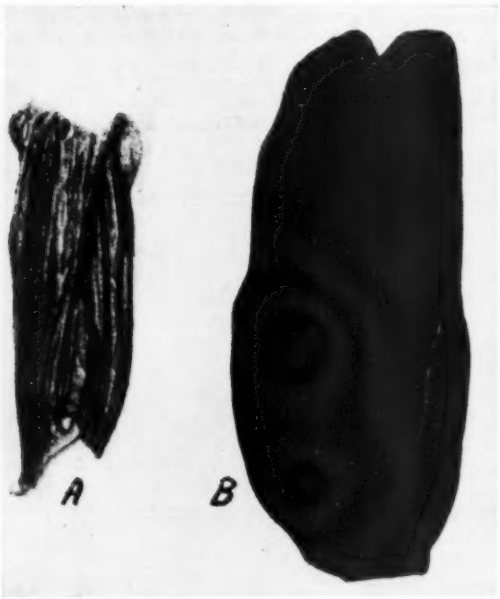

FIGURE II. Dog Number 13. Sartorius muscle two days after venous ligation (B), as compared with the normal muscle from the opposite limb (A). Intense œdema and cyanosis. (After D. Stewart Middleton.)

Again, let us consider what happens to a muscle from which the arterial supply has been cut off. Brooks found experimentally that the muscle shows a certain amount of necrosis and inflammatory changes, with later wasting, but that the fibrosis characteristic of ischæmic contracture does not occur. We have then to fall back on some other explanation, and Jepson's experiments on the dog, recorded above, would strongly suggest that stoppage of the venous drainage of a muscle would lead to typical ischæmic changes. To Middleton, of Edinburgh, must be given the credit of proving this thesis beyond all reasonable doubt. Working on the sartorius muscle of the dog, he completely abolished all venous drainage, while leaving the arterial supply intact. Rapidly, the typical tumefaction developed, and was followed by the characteristic fibrosis of ischæmic contracture (see Figures II, III, IV and V). The changes were due to acute venous congestion. This led to rapid swelling and the development of high tension in the muscle; muscle fibres were separated by a profuse effusion of lymph; pressure necrosis of muscle fibres occurred extensively, and finally the effused lymph became organized and the muscle became virtually a mass of scar tissue.

How does this explanation fit in with the clinical findings in cases of Volkmann's ischæmia in the forearm? A close consideration of the factors at work in these cases makes the same explanation complete.

The condition can follow most fractures of the forearm, but is associated most commonly with supracondylar fracture of the humerus in children under ten years of age. In this fracture the lower fragment is most frequently displaced backward, and the sharp, jagged, lower end of the upper fragment projects forward into the antecubital fossa, generally penetrating the *bracialis anticus* muscle (see Figure VI).

Great swelling occurs rapidly, and a hæmatoma forms in the antecubital fossa, the limited space which is already encroached upon by the projecting lower end of the upper fragment of the humerus. The space is bounded in front by the deep fascia, which prevents diffusion of the hæmatoma. The veins draining the muscle group comprising the *pronator radii teres*, flexors of the wrist, and superficial flexors of the fingers, pass through the inner side of the space and constitute a plexus, which, according to Middleton, can be covered by a sixpenny piece. The stage then is set for the development of ischæmia in this group of muscles.

FIGURE III. Dog Number 13. Two days after venous ligation. Microscopic section showing initial muscular degeneration and early invasion of the œdematous spaces by leucocytes. (After D. Stewart Middleton.)

If, in addition, forceful attempts at reduction of the fracture are made, and do not meet with success, then all the above factors are still at work, with the addition of a fresh access of bleeding into the antecubital fossa. One more factor is often added, and that is forcible flexion of the elbow to a very acute angle, and fixation of the arm in this position by the application of a bandage or of adhesive strapping which completely encircles the bent arm, and thus forcibly keeps up the tension in the antecubital fossa. The potency of this state of affairs as a factor in producing ischæmia is, of course, greatly accentuated if reduction has failed.

If an X ray plant is available, these deleterious factors are, unfortunately, not always allowed to rest on their merits. As a result

of X ray examination revealing forward displacement of the upper fragment, more force is rapidly administered, and I have known this to be followed by a third application of force within the first few days of the accident, without the achievement of successful reduction.

Regarding the symptoms and signs of Volkmann's ischæmia, the importance of prompt recognition of the condition cannot be too strongly stressed. The condition is quite amenable to successful treatment in its early stages, but a delay of twenty-four hours may result in disaster.

FIGURE IV. Dog Number 1. Sartorius muscle eight weeks after venous ligation. Almost complete replacement by fibrous tissue. (After D. Stewart Middleton.)

I would place in order of first importance the development of severe pain. The pain is agonizing; it is the pain of dying nerves, the pregangrenous pain with which we are all familiar. Such pain should not be. If the treatment of a fracture has been successful, the treatment should promptly relieve pain and make the patient comfortable. In addition to pain in the region of the fracture, the hand becomes swollen and dusky; the fingers cannot be extended; and any attempt to accomplish this movement increases the pain and causes the patient acute distress. Anæsthesia in the median or ulnar nerve area may develop and when this occurs, not having been present before attempted reduction of the fracture, the condition is very far advanced indeed, and may be beyond the reach of treatment.

Finally, I would mention absence of the radial pulse, which is commonly described as an important sign, but which I would view as a minor manifestation of ischæmia, and in some cases not of the gravest importance, particularly by comparison with the signs already mentioned. It must not be forgotten that minor degrees of ischæmia can, and do quite frequently, occur in the course of treatment of forearm fracture. A swollen, dusky hand and some pain on passive extension of the fingers call for prompt relief, or they may lead to inability to extend thumb or fingers completely after the fracture has finally united.

Prophylactic treatment is of vast importance. It is bound up with the methods of treatment of supracondylar fracture of the humerus, and

particularly with the frequently misunderstood and misapplied treatment of this fracture with the elbow in acute flexion, as advocated and practised with great success by the late Sir Robert Jones and his disciples.

The main prophylactic measure is undoubtedly early and complete reduction of the fracture. By achieving this objective, the surgeon at once lowers the tension in the antecubital fossa by the simple process of lessening its contents; the mass of the projecting lower end of the upper fragment of the humerus no longer projects into the fossa (see Figures VI and VII). Reduction before swelling becomes great is usually a simple process; but it can be really difficult when advanced swelling is present. In passing, it should be noted that reduction is not complete until the normal forward angulation of the lower epiphysis of the humerus is restored. This epiphysis points forward at an angle of about 35° with the shaft (see Figure VII).

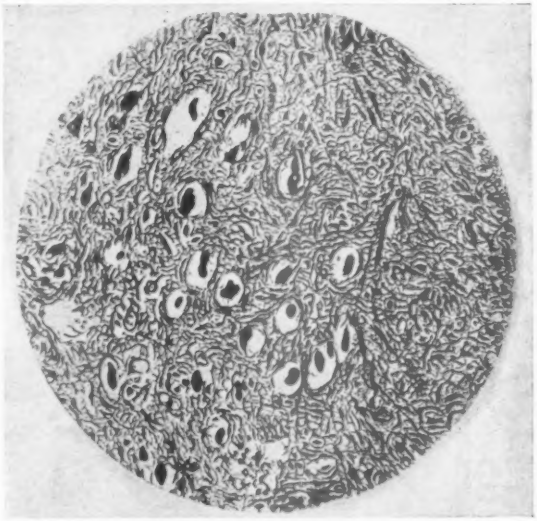

FIGURE V. Dog Number 1. Microscopic section of sartorius muscle eight weeks after venous ligation. Young cellular connective tissue and muscular remnants are to be seen. (After D. Stewart Middleton.)

I have found the method of acute flexion most satisfactory for retention. Before reduction is effected, it is generally found that the forearm cannot be flexed much beyond 90° without the use of force. After reduction, except where great swelling is present, the forearm can be readily flexed beyond 90° without its being necessary to overcome the slightest resistance; no force whatever should be used against tissue resistance; the movement of flexion should be stopped as soon as such resistance is encountered.

It is failure to realize this principle that induces surgeons to force the arm up into a position of complete flexion willynilly under the mistaken impression that Sir Robert Jones insisted upon it. The word acute, as here applied, means less than 90°, and was never meant to imply the position of maximum flexion. The only fixation needed is a

FIGURE VI. Supracondylar fracture of humerus before reduction. Note forward projection of lower end of shaft into antecubital fossa.

FIGURE VII. Same case after reduction, showing final result, treated in acute flexion with cuff and collar only for fixation.

cuff round the wrist, attached by a calico bandage to a collar around the neck. No splinting whatever is used, and the arm is left exposed and swinging freely without any fixation to the body or any obstructive clothing. The patient is encouraged to use the hand and fingers, and to attempt movement at the elbow from the beginning; above all, no encircling bandages or adhesive straps are put round the arm.

When great swelling is present, an attempt at reduction should be made with immediate verification of the position by X ray examination. If this attempt fails, I would advise three or four days' rest in bed with the arm elevated in a comfortable posterior plaster splint. It will then be found that the swelling has rapidly diminished, and complete reduction under X ray control can usually be achieved without the necessity of open operation.

When definite signs of ischæmia are present, the surgeon, bearing in mind that the limb as a whole matters more than the fractured bone, should set to work at once to combat the ischæmic condition. At this point I would stress, however, that I have never seen ischæmia develop in a case of supracondylar fracture when the fracture has been completely reduced and put up with the elbow in acute flexion. Nevertheless, if signs of ischæmia are present in a case of unreduced fracture, I would not advise a further attempt at reduction at once.

In mild cases of ischæmia it may be sufficient to remove encircling bandages, or splints, and, if the arm is in flexion at the elbow, to widen the angle of flexion and to elevate the limb. If the signs of ischæmia are advanced, and particularly if definite tumefaction is present in front of the forearm at the elbow, multiple incisions through the deep fascia, over the flexor and pronator group of muscles, should at once be made.

In late cases, when definite ischæmic contracture has developed, the clinical picture is very easily recognized. The forearm is wasted. It is held in a position of pronation due to contracture of the *pronator radii teres*. The fingers and thumb are contracted in flexion, but can be extended when the wrist joint is flexed, as this position allows the shortened and scarred flexor muscles of the finger and thumb to relax. In the worst cases the median and ulnar nerves are involved in the scarring, and anæsthesia and trophic changes are present in their cutaneous distribution, together with paralysis of the intrinsic muscles of the hand.

In many cases of established ischæmia, treatment can lead to great improvement, particularly if commenced early. Conservative treatment should always be tried first. It is based on the simple principle of gradually stretching the contracted muscles. The fingers and thumb are each separately fitted with anterior splints, applied with the wrist flexed so that the fingers can be held in extension at all joints. Over these splints a splint of the "cock-up" type is applied to the forearm and hand, projecting beyond the fingers.

This splint has a stem of malleable metal, connecting the forearm-piece with the hand-piece. At first the wrist is kept flexed, and gradually

from week to week the hand-piece is extended on the forearm-piece, with the fingers still splinted in extension. The splints are removed daily for the patient to receive the services of a good physical therapist. The application of heat is followed daily by assisted active movement, in which finger extension is encouraged. In this way marked improvement can be obtained with patience.

For cases in which conservative treatment fails, a number of operative measures have been devised, ranging from multiple tenoplasties to shortening of the radius and ulna, and excision of the elbow joint. Of these measures, I have had experience of only two, and can recommend them as worth while. I think Max Page, of London, deserves the credit of the first, and the second is a modification of it devised by Hamilton Bailey, of Birmingham. Max Page separates the origin of the flexor pronator group subperiosteally from the front of the internal epicondyle, and by extending the wrist and fingers slides it down and allows it to secure a fresh attachment in the forearm. Hamilton Bailey suggested completely removing the inner epicondyle with its attached muscles, and suturing it to the ulnar shaft lower down.

In established congenital wry-neck, excellent results are obtained by completely dividing both heads of origin of the muscle, together with any contracted bands of cervical fascia. The operation is followed by a course of active exercises under the direction of a physical therapist.

The results in my experience have been equally good when a plaster cast was applied to hold the head in the corrected position after operation, or whether this measure was not resorted to, but corrective exercises were adopted earlier. Patients with congenital wry-neck are seldom referred to a surgeon for treatment in the acute stage of tumefaction of the sterno-mastoid muscle. I have thus had no experience of treatment in the first twenty-four hours after birth. Considering the pathology of the condition, however, I should think in a case seen as early as this, or even within the first few days, multiple incisions in the deep fascia over the lower part of the sterno-mastoid muscle would be worth while.

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EXPERIMENTAL OBSERVATIONS ON INCREASED INTRACRANIAL PRESSURE.

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Introduction.

The study of reactions to increased intracranial pressure is simplified if a general uniform increase of pressure is used. By direct injection of physiological saline solution into the cranium, probably into the subarachnoid space, Cushing[1] freed his experiments of the objections of shearing and indeterminable pressures applicable to the use of a balloon inflated under the *dura mater*; this latter method had been used by previous experimenters. He demonstrated that a rapid rise of general intracranial pressure produced no manifest reactions of the neuro-cardio-vascular control until the intracranial pressure approximated the systolic blood pressure. By the use of a similar method Dixon and Halliburton[2] confirmed these results. In his paper Cushing notes that emptying of the cerebral veins does not take place, but that the capillaries may empty when intracranial pressure is greater than blood pressure. Wolff and Forbes[3] later demonstrated that while the intracranial pressure is rising to arterial blood pressure, the diameter of the pial vessels becomes wider than it was at the commencement of the experiment. Wolff and Blumgart[4] demonstrated that over this period the velocity of cerebral circulation was reduced, and Gibbs[5] showed that the minute-volume was reduced.

Cushing studied the mechanism of this alteration of blood pressure by vagotomy and section of the spinal cord, and concluded that the initial fall of blood pressure was due to direct stimulation of the vagal centre, and the subsequent rise was effected by the stimulation of the para-vertebral sympathetic system from the medulla. That these effects may have been initiated from the carotid sinuses was disproved by Heymans,[6] but he gives little detail of the actual changes which took place when the intracranial pressure was raised in animals in which the carotid sinuses were denervated. Guernsey, Weisman and Scott[7] found that after denervation of the carotid sinuses and aorta, if the intracranial pressure was raised slowly, the blood pressure rose more readily and to a higher level than before such extirpation.

Though Cushing noted that apnœa may supervene when the intracranial pressure approaches the systolic blood pressure, he did not

further analyse the mechanism producing this result. Eyster[8] expressed the opinion that apnœa resulted from the vulnerability of the respiratory centre to anoxæmia, though he did not definitely prove that the centre was at this time insensitive to stimulation. Schmidt[9] raised the intracranial pressure above systolic blood pressure for about thirty seconds and suddenly lowered it; apnœa resulted. This was considered to be due to the flushing of the respiratory system with arterial blood, and to result from functional inactivity due to absence of the usual stimuli. In an earlier paper[10] he deals with apnœa resulting from absence of oxygen in the respiratory centre or the inability of the respiratory centre to use oxygen.

Leonard Hill,[11] Becht[12] and Weed and Flexner[13] have demonstrated that the pressure in the torcular Herophili does not rise to the height of the intracranial pressure when this is raised by injection of fluid, but continued to regard the torcular pressure as a true index of the cerebral venous pressure. Leonard Hill[14] had previously demonstrated the free communication between the sagittal sinus and general venous system under normal conditions of intracranial pressure. Cushing,[15] observing the sagittal sinus through a window, noted that it collapsed when intracranial pressure was raised; the fact that he had already incised the *dura mater* probably led to error, as will be noted later.

The effect of raising the intraspinal pressure above systemic blood pressure has been found by Montgomery and Luckhardt[16] to cause first exaggeration and then failure of the knee jerk reflex.

Lambert Rogers[17] has stated that brain tissue is easily compressible, but it was shown by Grashey[18] that the coefficient of compressibility of brain tissue is slightly less than that of water.

In all the investigations in which the intracranial pressure was raised quickly, either instantaneously or over a period of a few minutes, a system of subarachnoid injection was used; this gives a uniform general increase throughout the subarachnoid and ventricular systems (Massermann and Schaller[19]).

SCOPE OF INQUIRY.

It becomes apparent from this review that the major fields of inquiry into physiological effects of rise of intracranial pressure are: (i) anatomical and physical factors presented by the intracranial vasculature; (ii) observation of behaviour of intracranial vasculature under conditions of increased pressure; (iii) observation of reactions of cardio-vascular and respiratory mechanisms; (iv) consciousness and more simple central nervous system reflexes.

In commencing the study of reaction to increasing intracranial pressure it was decided to investigate reactions to a general uniform increase in intracranial pressure. The field was further limited by the regulations governing anæsthesia in the use of laboratory animals. It

was, therefore, to changes in the general and intracranial vasculature and respiratory function that the present study was restricted. Observations relative to state of consciousness could not be carried out.

ANATOMICAL AND PHYSICAL CONSIDERATIONS.

The brain *minus* its vasculature may be considered as a liquid in an almost rigid container. So, a uniform increase of intracranial pressure can affect only the vapour pressure of the solvent and the degree of the possible saturation of fluid-gas solutions. Since there is no evidence that the former factor plays any part in the physiological activity of the nerve cells, and since in the latter state it is the actual amount and differential pressure of the gas which are the known factors of importance, it is improbable that uniform increase of pressure on the nerve cells can have any direct effect which would excite abnormality of function.

The blood vascular system, however, has a differential of pressure from one type of vessel to the next in sequence. It is on such a system of collapsible tubes with a gradation of pressure that one would expect the major effect of a uniform increase of pressure to be most evident. Secondary disorder of the neuronal nutrition would result from such interference with vascular flow.

In association with the circle of Willis, the major arteries branch off to the nervous system. These and most of the larger branches lie in the subarachnoid space in which the origin of the smaller arteries is situated. These then enter the brain substance and divide eventually via arterioles to capillaries; the venules from these join to emerge at the brain surface into larger venous trunks. In the subarachnoid space many capillary junctions between arterioles and venules are present. Thus all of these vessels are subjected directly or indirectly to the pressure which is in the subarachnoid space. From the subarachnoid space the larger veins pass into the dural sinuses, traversing both arachnoid and dura at this spot, and form narrow slit-like channels through these membranes.

The dural sinuses have a noteworthy arrangement relative to the supporting tissues. Those over the lateral convexities run in depressions in the bones, for example, the spheno-parietal and sigmoid sinuses; but the other great sinuses all lie at the junction of three almost inextensible folds of *dura mater*, and, as the falces or tentorium are tightly drawn and firmly attached, it is evident that intradural pressure, while applying directly to the cerebral and arachnoid veins, has an entirely different relationship to the dural sinuses. While an arachnoidal vein, if full, must always be at a pressure equal to or above that of the cerebro-spinal fluid pressure, it is quite possible that the dural sinuses may be at a pressure lower than this. In the absence of valves between the sinuses and the arachnoid veins there must be, when circulation is progressing from the latter to the former, a pressure gradient in favour of the arachnoidal veins.

The most outstanding physical consideration of circulation through a system of collapsible tubes, subjected to an external pressure below the maximum pressure in this system, is that circulation is maintained with little, if any, diminution in calibre of vessels, except just at the point of outflow. The variation of differential pressure between the inlet and the outlet governs the alteration of volume flow per minute. This happens because, with increase of perivascular pressure, there is a compression valving at the lower pressure outflow; if the tubes contain incompressible liquid surrounded by incompressible liquid under pressure in a rigid container, all the vessels toward the higher pressure in the tube system immediately take up a pressure from the higher pressure tubes sufficient to overcome this valving, and so the effective circulating pressure becomes the difference between the highest available pressure at the inlet and the pressure surrounding the tube system.

However, if at any point there is a constriction, the velocity of circulation through this constriction is increased; and, according to Bernoulli's theorem, if velocity increases, pressure is reduced. It becomes evident that a point of constriction in a simple physical system is adjacent to a point of low pressure, and in practice it is found that, starting from such a point, collapse spreads toward the lower pressure tubes and empties them completely. Then they fill slowly through the constriction at a pressure a little above the surrounding pressure, until the outlet is reached, when collapse starts again at the constriction and the low pressure system empties rapidly. So, for a short time, such an area is completely collapsed and slowly refills. Though at first thought it might appear that the arterioles, constituting the peripheral resistance, might show such a constriction, newer conceptions of peripheral resistance lay equal emphasis on all the small vessels. When it is realized that resistance to flow of a liquid is inversely proportional to the fourth power of the radius (r^4) of the vessel concerned (Poiseuille's law), it can be readily seen that a slower velocity of circulation can occur through these small vessels than through the artery, but yet the resistance to the flow be increased. From the point of view of hydrodynamics no constriction is present; in fact, a wider vascular bed is here provided.

So far, these vessels have been considered as simple collapsible tubes. That they show muscular reaction has been denied and affirmed. So minute are the changes noted in the latter observations (Forbes, Finley and Nason[20]) that for all practical purposes it can be assumed that no spontaneous neuro-vascular control is present, and that the problem remains one of simple hydrodynamics.

EXPERIMENTAL METHODS.

Dogs were used. They were given 0·06 gramme (one grain) of morphine and kept under light anæsthesia with chloroform. Blood pressure was recorded by mercury manometer from the femoral artery. In the course of the tubing a needle of the same length and bore as that in the *cisterna magna* was interpolated; this allowed comparison

between the amplitude of fluctuation in the artery and the cranium. Respiration was recorded by a tambour connected to a rubber bulb lightly compressed by a band of gauze fixed round the animal's thorax.

Intracranial pressure was recorded by a mercury manometer, on the same base line as blood pressure, connected to a wide bore needle introduced through the atlanto-occipital membrane. With the animal's head flexed, this manœuvre is easily accomplished. In no case did it cause damage to the medulla, and it gave an almost completely water-tight junction and reduced operative trauma to a minimum. Through this needle the intracranial pressure was raised by introduction of Ringer's solution (sodium chloride 0·9%, potassium chloride 0·003%, calcium chloride 0·002%). In the descriptions of experiments previously mentioned two factors are overlooked. In all of them the Ringer's solution must have regained atmospheric temperature before it reached the cisterna; the large vessels and long tubes used to raise the pressure by simple elevation buffered all the fluctuation of the intracranial pressure due to arterial pulsation. Both these features were corrected by using a needle with a side tap. A was connected to the manometer and B to the pressure bottle containing Ringer's solution at 38° C. When it was desired to raise the intracranial pressure, the two-way tap was turned to connect B and C, and fluid was run through until warm solution arrived; then B was connected to D. If accurate estimation of fluctuation was desired, the side tap was turned completely off; if a constant pressure was required it was left in direct connection between pressure bottle and the *cisterna magna*. The slowly infusing fluid was then rewarmed by the vasculature of the muscular tissue through which the needle passed.

So that the amount of fluid injected could be measured, the pressure bottle was made to include a separate compartment from which fluid could be used and measured to 0·1 cubic centimetre. For warming purposes the fluid was taken from the large compartment of the bottle; for injection purposes the fluid was taken from the graduated measure, which could be replenished by simply tipping the bottle while clamps were on both tubes.

The pressure at which the blood was present in the sagittal sinus was recorded by a tambour connected to a needle in the sagittal sinus before this enters the bony canal at the occipital protuberance. For these experiments the animal was hirudinized with the extract of forty leech heads. Though this manometer method gives a slight lag in record, the needle used was chosen so that there would be no obstruction of circulation, and it is probably the only method applicable to the membranous part of the sagittal sinus as opposed to the bony portion.

The actual flow of blood in the veins was recorded in hirudinized animals by fitting a cannula into the sagittal sinus in the membranous portion. The blood was collected in a Conder's electric tipper (set for five centimetres) and introduced back into the femoral vein.

The intracranial vessels were viewed directly through a window screwed into a trephine opening in the skull after the *dura mater* had

been divided. First the outside shell A was screwed firmly into the trephine hole, with some tow in the thread to render the junction watertight. This shell was filled with Ringer's solution and all air bubbles were removed. The glass window was then placed in position and the inside shell was screwed firmly down. Usually the parietal region was chosen, but in special cases a field including the sagittal sinus was used. Observations were made through a Zeiss binocular dissecting microscope, giving a magnification of 31. To escape glare from the window into one or other objective it was often necessary to incline the window and microscope and to pour a thick film of water onto the window glass. All the reflected light was then cast onto the blackened inside of the inner shell and the image of the vessels was not materially distorted.

The relation between the arachnoidal venous pressure and the sinus pressure was studied by the use of a window in the skull, and the records were given from the sagittal sinus pressure.

It is a fair assumption that, if the arachnoid veins are full of blood with a sinusward direction of circulation, the pressure in the arachnoid veins is at or above that of the surrounding fluid, that is, the intracranial pressure.

The patency of the sinus under pressure was investigated by opening the sinus at the anterior end in the recently dead animal. Intracranial pressure was then raised to 200 millimetres of mercury, and a coloured fluid was injected into the external jugular veins at the post-glenoid foramen at 20 centimetres of water pressure.

The effect of carotid sinus reflexes on the changes produced was investigated by denervation and stimulation. The sinus reflexes were stimulated by Marshall's method[1] by intravenous injection of 0·15 cubic centimetre of N/50 potassium cyanide per kilogram of body weight. This in the normal animal excites a marked hyperpnœa and slight rise of blood pressure. Denervation was accomplished by clean dissection of the common, internal and external carotid arteries for at least one centimetre beyond the bifurcation. Not until there was no hyperpnœa to the cyanide test was it considered that denervation was complete.

In some experiments it was necessary to remove the efferent effects of the vagus nerve on the heart, but yet to be able to reintroduce these effects later. For this purpose the nerves were laid on clean brass tubes through which 10% sodium chloride solution cooled to –2° C. was circulated. Stimulation above the tube was used as a control of complete blockage. Simply warming the nerves to 37° C. caused instant recovery of function.

Compression of the spinal cord was effected by ligation and division of the cord and *dura mater* between two ligatures at the level of the seventh cervical vertebra. The needle was then inserted into the lower spinal dura and the pressure was raised in the usual way.

[1] Personal communication.

The respiratory centre was stimulated by two methods. Reflex excitation from the carotid sinuses was effected by intravenous injection of cyanide. Direct stimulation was given by connecting the terminals of a faradic coil to the *cisterna magna* needle and an artery forceps on the sterno-mastoid muscle. Though great diffusion must have occurred, yet an efficient stimulation of respiration was obtained.

OBSERVATIONS.

Raising of Intracranial Pressure, with Window Observations of the Intracranial Vasculature.

Twelve animals were used, and twenty-eight observations were made on raising the intracranial pressure; the time taken to raise the intracranial pressure to the same level as systolic blood pressure varied from three and a half to forty-five minutes.

When viewed by a window inserted in the skull, the only discernible movements of the vasculature at normal intracranial pressure were streaming of granular blood content in some capillaries and venules and the winding and unwinding movements of arteries. The adjective granular is used because it is realized that, with the magnification employed, the definite lack of homogeneity of the blood in these vessels must have been due to the presence of aggregates of corpuscles, not to the perception of individual corpuscles. With the binocular microscope no constriction at the junction of arterial branches with the main stem is apparent. This phenomenon, recorded by Forbes, is seen only with monocular microscopes, and is, in fact, an illusion.

When the pressure of the cerebro-spinal fluid was increased no significant diminution of calibre of the small vessels occurred until a level a little below that of the systolic blood pressure was reached, when the arteries showed an obvious decrease in the normal movement. Up to this level of pressure no definite collapse of veins occurred, except near the sinuses, where, from the commencement of rise of the intracranial pressure, a cuff constriction appeared, with intermittent emptying of the venous content into the sinuses. This took place most usually just after inspiration.

During the rise of intracranial pressure to this level two features of equal significance relative to the absence of changes in calibre are manifested. The first concerns the colour of the blood in the vessels. Normally the colour distinction between the artery and vein, though definite, is not that seen in other vessels; the venous content is not the deep blue plum colour of reduced hæmoglobin, but only a few shades off oxyhæmoglobin. From Schmidt's data[10] the calculated degree of reduction of the hæmoglobin is 7%. However, as the pressure slowly rises the colour difference becomes gradually more obvious, until, when the difference between the intracranial pressure and the mean blood pressure is about 30 millimetres of mercury, the venous content is a deep purple with the blue element extremely well marked. The quick gradation

from the red of the artery to the blue of the vein, when seen along the capillaries, is most striking as the pressure rises.

The second important feature is the visibility of a granularity of the blood in some of the small capillaries and venules at normal levels of pressure and rate of circulation. As the pressure of the intracranial fluid rises this becomes visible in the larger veins until only the major intergyral veins fail to show it. It does not appear in the arteries until the intracranial pressure has almost reached the systolic arterial pressure and the circulation in the arteries practically ceases. From the rate of movement of this blood, which is discernible because of the granularity, the rate of circulation is readily assessable. It would appear also that the actual presence of granularity indicated a slow flow of blood. As the pressure slowly rises the movement of the blood becomes gradually slowed, until, as the intracranial manometer enters the lower levels of the diastolic blood pressure, the blood in the capillaries and venules becomes almost stationary; a sluggish, intermittent movement only of the granules is seen. Granularity which is now present in the larger veins, shows a slower movement than before, but usually a greater velocity than in the small vessels.

Only when the intracranial pressure is within a few millimetres of the systolic level do the small vessels collapse; the blood does not empty quickly onto the venous side, but more rapidly into the smaller arterioles, which collapse, showing only occasional granules of red corpuscles along them; where they join the larger arterial branches a to-and-fro movement, synchronous with the pulse wave in the larger arterial branches, is seen. At this pressure level, effective circulation in the cerebral capillaries ceases. In the greater veins there is a continued slow forward movement of the blood; this has been observed to continue for half a minute with the intracranial pressure 10 millimetres of mercury above the systolic blood pressure level recorded from the femoral artery.

When a fall in the intracranial pressure is allowed, it requires considerably greater difference in pressure before the reversal of these processes occurs. As much as 20 to 30 millimetres of mercury difference may be necessary before the arterioles and capillaries refill from the arterial side. It is probable that this phenomenon is in conformity with the law that resistance to passage of fluid through tubes varies inversely as the fourth power of the radius; here the arterioles are of negligible diameter at the commencement of filling, and as no active expansion can take place the resistance to filling is enormously increased. When this replenishment has been effected, forward movement of the blood occurs in all capillaries and veins, at first slowly, with marked granularity and blueness in the great veins, but rapidly accelerating until, when the pressure has fallen to its original level, the state of the vessels is the same as at the beginning.

In two cases it appeared that the brain was hyperæmic when the pressure had been allowed to recover its normal level for five minutes. Though no vessels other than those seen at the commencement of the

experiment were visible, and no increase in calibre of those previously seen could be determined, the area appeared redder with less contrast between the arteries and veins than at the beginning. It is possible that this was an error due to contrast to the state during raised pressure; if so, it should have occurred in all cases. In three experiments the small vessels failed to open synchronously; some remained closed even until the intracranial pressure was within 10 millimetres of mercury of normal. Resistance to inflow in relation to the patency of the other vessels probably influenced this.

The anterior point of the sagittal sinus was exposed by trephine in six dogs. In three a window was fitted without incision of the *dura mater,* in three the *dura mater* was incised parallel to the left edge of the sinus before the window was fitted. In those in which the *dura mater* was intact there was slight fluctuation of the sinus with respiration. When the intracranial pressure was raised no compression of the sinus was seen, nor was collapse present. The window was removed and the pressure was again raised. In this case, when the intracranial pressure approached systolic arterial blood pressure, the sinus collapsed in exactly the same manner as when the carotid and vertebral arteries were occluded. With the window in the skulls of those animals in which the *dura mater* was incised the sinus collapsed under pressure exactly as described by Cushing, in marked contrast to the arachnoidal veins, which remained full and distended with blood up to the edge of the sinus.

The pressure of blood in the sagittal sinus may rise a little (four to five centimetres of water) at the commencement of raising the intracranial pressure, but rapidly falls to a low level (in one case to –6 centimetres of water), and remains there so long as the compression is kept up, if respiration is maintained. Prolonged periods of apnœa cause a marked rise (to 25 centimetres of water) of this pressure, reflecting the rise in venous pressure throughout the body. When intracranial pressure is released the pressure in the sinus slowly rises above the original level, even to 30 centimetres of water, from which level it slowly falls to normal over a period of ten minutes or more (Figure I).

The flow of blood runs approximately parallel to the pressure. A decrease in flow was discernible as the pressure was first raised, with gradual reduction until complete cessation occurred with the intracranial pressure above the systolic arterial blood pressure. When the pressure is released a reversal of this change occurs (Figure II). The alteration in colour of the venous blood previously noted is very apparent in the sinus blood.

In seven dogs, recently dead, with an opening over the anterior part of the sagittal sinus, Berlin blue solution was introduced through the right external jugular vein at 20 centimetres of water pressure. This flowed readily through a small incision at the posterior end of the exposed sagittal sinus, and continued to do so even though the intracranial pressure was raised to 300 millimetres of mercury.

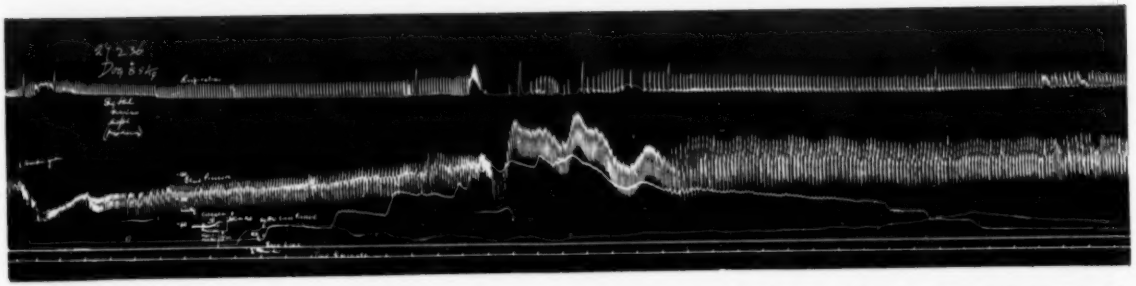

FIGURE I. Dog, 8·5 kilograms. Morphine and chloroform. The record shows respiration, blood pressure and intracranial pressure in millimetres of mercury, sagittal sinus pressure in centimetres of water, and time half minutes. The hirudin contained depressor substances and the initial fall of blood pressure and subsequent rise during the early stages of compression are due to these. The blood pressure and respiratory changes are otherwise typical of the changes seen in the normal animal during compression. After decompression the blood pressure shows an unusual range of fluctuation. The sagittal sinus pressure, except in the first stage of the elevation of intracranial pressure, shows an inverse relationship to the intracranial pressure.

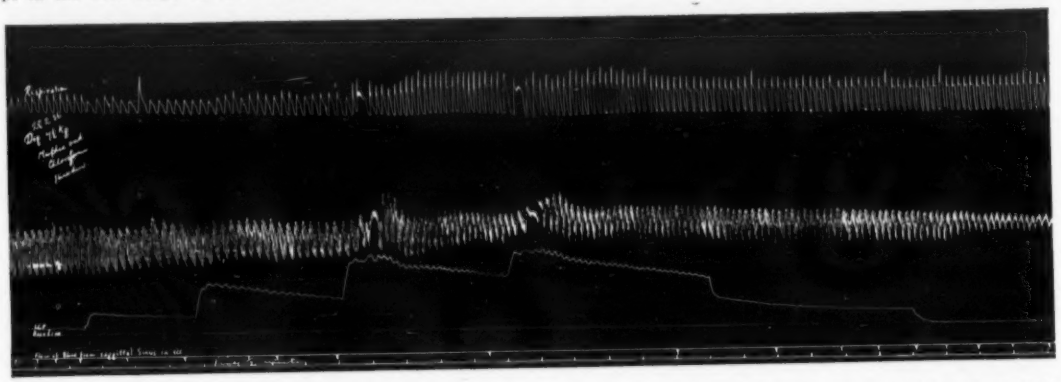

FIGURE II. Dog, 7·5 kilograms. Morphine and chloroform. In addition to respiration and intracranial and arterial blood pressure, the flow of blood from the sagittal sinus is shown as 5 cubic centimetre amounts. The proportion of this to intracranial pressure shows an inverse relationship.

While considering the behaviour of the intracranial vasculature, it is worthy of note that, though two to four cubic centimetres of solution are usually required to raise the intracranial pressure quickly to 120 millimetres of mercury, in two dogs one cubic centimetre only produced such an elevation.

While the intracranial pressure was being raised, the fluctuation of the intracranial pressure manometer gradually increased, synchronously with the pulse in the arteries, until the blood pressure was reached. The amount of fluctuation varied greatly; in some cases a few millimetres only at the maximum, in others a pressure of 10 to 20 millimetres of mercury was recorded, that is, one-third of the pulse pressure recorded from the artery.

Reactions of the Cardio-Vascular System.

Alterations of Pulse Rate and Blood Pressure.—The most remarkable feature of the blood pressure tracing was the absolute stability of the pulse rate and blood pressure until the intracranial pressure approached systolic blood pressure level. The intracranial pressure was retained at 10 millimetres of mercury below diastolic blood pressure for thirty minutes in three animals, all of which failed to show any rise of blood pressure. This was so whether the vagus was frozen or intact; the integrity or otherwise of the innervation of the carotid sinuses did not have any influence on this stability. When the intracranial pressure was elevated rapidly to or above the systolic blood pressure level, the pulse rate became slowed; in eleven cases so marked was this slowing that a lowering of blood pressure usually of twenty millimetres of mercury occurred. In three cases the blood pressure fell forty to fifty millimetres of mercury, and in one heart action ceased for ten seconds. This fall of blood pressure resulted in an equal fall in the intracranial pressure.

Cases of complete vagus inhibition of the heart recorded by Cushing[1] were not found to occur so long as the temperature and concentration of the Ringer's solution were carefully maintained.

After a period of ten to fifteen seconds, in all cases the blood pressure rose steeply to a pressure at least twenty, usually forty, millimetres of mercury above the level of intracranial pressure. In all cases during this rise of blood pressure the pulse rate was slowed to about two-thirds of the original pulse rate, and the pulse pressure was correspondingly increased.

In five experiments this rising blood pressure was "chased" by the intracranial pressure; in these cases the blood pressure continued to rise above it. When (thirteen experiments) the intracranial pressure was allowed to drift downward after the blood pressure had risen, the latter followed it, twenty to forty millimetres of mercury above it, following it closely and even reproducing artificially produced major curves of the intracranial pressure upward (Figure III). When the blood pressure reached a level, except in three cases, at least ten, sometimes twenty, millimetres of mercury below normal, the extreme slowing

of cardiac action ceased, the pulse rate increased suddenly to the pre-compression level and the blood pressure came back to a normal level, but with marked respiratory influence on the tracing due to the slower, deeper breathing which had commenced. In the other three cases, when the blood pressure reached the original level, the slowing of pulse ceased suddenly after remaining two to three minutes at the normal pressure level.

Seven experiments were carried out with a very slow rise (over forty minutes) of the intracranial pressure to the blood pressure level. In these cases no initial vagal effects were detected. In two dogs the blood pressure rose steadily thirty millimetres of mercury above the

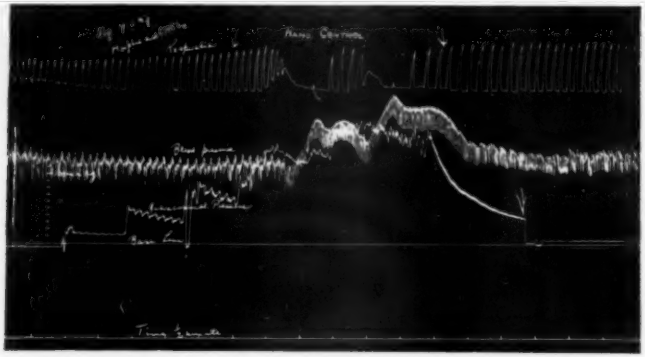

FIGURE III. Dog, 7·5 kilograms. Morphine and chloroform. The intracranial pressure was raised in steps. No change of blood pressure occurred until the intracranial pressure exceeded the blood pressure. In this interval the respiration became deeper and showed a typical onset of apnœa at the time of alteration of blood pressure. After the rise of blood pressure and recovery of respiration, the intracranial pressure was raised again. A further rise of blood pressure occurred associated with apnœa. The intracranial pressure was maintained and then allowed to fall, and at the large arrow the cranium was decompressed. Sudden recovery of the precompression pulse rate is shown 34 seconds later. The deep slow breathing typical of the post-compression period is present, and the intracranial pressure is stabilized 16 millimetres of mercury above the precompression level and shows marked respiratory influence.

intracranial pressure to a level of 240 and 200 millimetres of mercury after fifty-five and sixty minutes respectively. At these points cardio-vascular failure occurred associated with irregular respiration. In the other four dogs, when the blood pressure was disturbed by the rising intracranial pressure, cyclical rises and falls of blood pressure, similar to those described by Cushing and Eyster, and associated with cyclical breathing, commenced. The other experiment is seen in Figure IV.

Nervous Control of Cardio-Vascular Reactions.—Consideration of the problem of the nervous control of cardio-vascular reactions may be divided according to the "time-height" relationship of the intracranial

pressure: (*a*) When the pressure is rising to systolic blood pressure. (*b*) When it is maintained at such a height that the blood pressure remains raised above its normal level. (*c*) When the intracranial pressure is falling to its normal level.

It was considered possible that the failure of the blood pressure to rise in the first period might have been due to some nervous control of the cardio-vascular mechanism. It was considered most likely that this would, if present, be of an inhibitory nature. As most inhibitory phenomena are effected via the vagus, experiments were carried out raising the intracranial pressure with the vagus nerve cooled to cut off afferent or efferent vagal impulses (five experiments). When the vagus

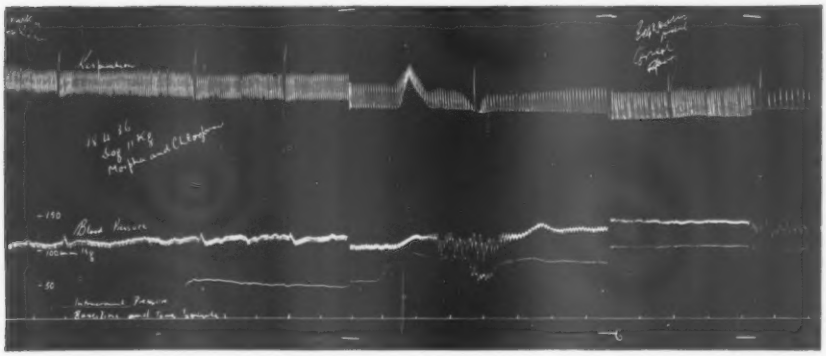

FIGURE IV. Dog. Morphine and chloroform. The intracranial pressure was raised slowly to the blood pressure over a period of twenty-five minutes, and then retained just below it. The blood pressure and pulse rate remained steady for another ten minutes, and then began to show irregularity with an increase of these phenomena, until the animal died fifty minutes after the commencement of compression. At the thirty-five minute time the pupils were small, the corneal reflex was present, and the eye was bulging almost from its socket. Ten minutes later the pupil was dilated, the corneal reflex absent and the eye protruding between the lids (see Figure VII).

was cooled the pulse rate increased immediately 20 to 30 per minute, varying in different animals, the systolic blood pressure rose slightly (up to 40 millimetres of mercury), with a rising of diastolic pressure out of proportion to systolic increase; for example, in experiment 1 the systolic pressure rose 10 millimetres of mercury and the diastolic pressure 36 millimetres; in experiment 2 the systolic pressure rose 40 and the diastolic pressure 60 millimetres of mercury. When in these preparations the intracranial pressure was raised, no further significant rise of blood pressure occurred until the intracranial pressure was 10 to 20 millimetres of mercury below systolic blood pressure.

Then in the second period, without preliminary falling, the blood pressure began to rise without any definite slowing of pulse rate, but with increased pulse pressure. When a level 30 to 40 millimetres of

mercury above intracranial pressure was reached the blood pressure ceased to rise, and maintained that level with increased pulse pressure, for example, an increase from 14 to 40 millimetres of mercury. In one experiment, while this plateau was being maintained, the blood pressure rose suddenly somewhat over 70 millimetres of mercury, accompanied by an irregular slow pulse rate. In all other instances it maintained a level in relationship to the intracranial pressure.

When the intracranial pressure was returning to normal, in all cases the blood pressure was found to remain at a level somewhat above the precompression level, for example, 180 millimetres of mercury from 140 millimetres of mercury, which was immediately reduced to the pre-freezing level by restoring the vagal function. When this was effected suddenly, an instantaneous fall of blood pressure and very slow heart action occurred to a level below normal; a gradual rise of pressure to about precompression level followed this syncope; in no way did the features of return of vagus function differ from those occurring in dogs which had not been subjected to increased intracranial pressure.

In order to determine whether any of the vagal effects set out in the first part of these records, and further amplified by freezing, may have had their sensory origin in the carotid sinuses, the intracranial pressure was raised in animals in which these organs had been denervated. Six experiments were carried out on three animals. In all cases the blood pressure showed no significant change until the intracranial pressure approximated the systolic blood pressure. This was followed in four experiments by a marked fall in blood pressure, with slowing of the pulse rate and blood pressure, for example, from 68 to 42 pulse rate and from 190 to 50 millimetres of mercury systolic blood pressure, which was followed by a rise of blood pressure slower than in the intact animal and with a slowed pulse rate, until the blood pressure was from 10 to 20 millimetres of mercury above intracranial pressure. In the two other experiments on the same animal, in which an initial fall of blood pressure occurred on increasing intracranial pressure before denervation of the carotid sinuses, this did not occur; but with a slowed pulse rate an immediate rise of blood pressure to 20 millimetres of mercury above intracranial pressure occurred. In all experiments, while the level of intracranial pressure producing a rise of blood pressure was held, a differential of pressure varying from 10 to 20 millimetres of mercury was maintained in favour of the blood pressure. When the intracranial pressure was allowed to fall, the blood pressure and pulse rate quickly regained the precompression level and rate, without any sudden transition from slow to fast, as seen in the intact animal.

In two animals in which the spinal cord was cut, increase of intracranial pressure produced vagal effects only without rise of blood pressure and consequent complete apnœa. When the cord below the section was compressed no effect on the blood pressure was produced.

In two dogs artificial respiration was carried out by intratracheal insufflation from a pump, while the intracranial pressure was raised to

400 millimetres of mercury (not recorded on manometer). The blood pressure rose to a level above 300 millimetres of mercury before dropping abruptly at the end of two minutes. Complete apnœa was present throughout. When the intracranial pressure was lowered, both the vascular and respiratory centres recovered.

Respiratory Reactions.

Phenomena Observed.—The value of these observations is greatly limited by the fact that apparatus to record the respiratory exchange of the animal was not available. The results are thus limited to records of rate and relative depth of respiration in each case.

During the period when the intracranial pressure was being elevated to intracranial pressure level, a slight increase in depth of respiration without increased rate was noted in most cases. In none was there depression of respiration. In one case only was there a marked hyperpnœa when the intracranial pressure became equal to the blood pressure. Before compression, however, this animal showed irregular breathing at the rate of 80 per minute.

In all cases, within fifteen seconds of the intracranial pressure's attaining such a point in relation to blood pressure that sharp alteration in blood pressure resulted, apnœa occurred, lasting from twenty seconds to three minutes; and in no case did breathing recommence until the blood pressure had increased to a level above the intracranial pressure for at least fifteen seconds, usually thirty seconds, sometimes three minutes. The manner of onset of the apnœa appeared to be different in each animal: in one the amplitude of respiration gradually decreased over six cycles to apnœa in complete expiration; in another with three or four cumulative inspiratory efforts the chest became fully expanded, with gradual emptying during the period of apnœa. The other animals showed a degree of inflation at the beginning of apnœa varying between these extremes.

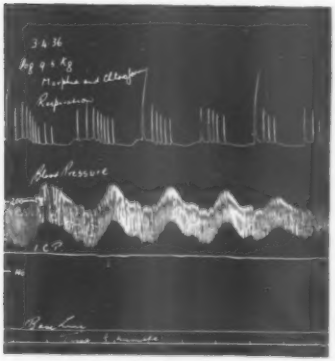

FIGURE V. Dog, 9·5 kilograms. Morphine and chloroform. The intracranial pressure was raised over a period of thirty-five minutes. Shortly after the blood pressure began to rise, the respiration and blood pressure showed cyclical changes which persisted until the death of the animal forty-five minutes later. The main feature of interest is the commencement of respiration at the period of lowest blood pressure.

When the blood pressure rose above the intracranial pressure the animals in two cases took a series of deep breaths, followed by respirations of less amplitude. In all other cases, and in these after the first few deep cycles, the respiration rate was much slower than before cerebral compression; the amplitude was increased greatly. In the case observed longest

(thirty minutes), after a single compression of six minutes' duration (apnœa one minute), the respiratory rate (twenty-two per minute) was slower and the amplitude greater (1·2 centimetres) than before compression (thirty-two per minute and 0·7 centimetre respectively).

In the four cases showing cyclical breathing each was of different type, corresponding to those described by Eyster in general outline, but differing in one essential: the onset of the cycle of breathing was not coincident with the peak of the blood pressure rise, but occurred half a minute after it, and so corresponded in point of time to the trough of the blood pressure wave. Apnœa then occurred before the subsequent rise of blood pressure (Figure V).

The Nature of the Apnœa.—In those animals with controllable vagotomy, though the type of respiration is altered by vagotomy, those features which occur in the intact animal are manifested. In the animals in which the carotid sinuses were denervated the same features were present as those in the animals with intact sinuses. In two animals subjected to cyanide injection during the period of apnœa no response whatever occurred. In another experiment the cyanide was injected twenty seconds after the commencement of apnœa, but with intracranial pressure below blood pressure. No effect was produced for forty-five seconds, when a marked hyperpnœa occurred, which gave place to apnœa when the intracranial pressure was again

FIGURE VI. Dog, 8 kilograms. Morphine and chloroform. Transpose respiration fifteen seconds to the left. The sensitivity of the normal respiratory centre to impulses from the carotid stimulated by cyanide is shown in contrast to the insensitivity during compression apnœa. The marked hyperpnœa which develops as soon as the respiratory centre recovers, is also striking. An incidental observation is the rise of intracranial pressure 6 millimetres of mercury which follows administration of cyanide.

elevated above the blood pressure (Figure VI). In two other dogs the medulla was stimulated via the *cisterna magna* needle. Though the stimulus in the precompression period gave a very pronounced hyperpnœa, during the period of apnœa no response whatever was elicited.

Incidental Observations.

In all those (six) animals in which the intracranial pressure was slowly raised, and in that in which the intracranial pressure was maintained just below blood pressure for one hour, proptosis and rhinorrhœa occurred. The eyes were noticeably protruded after a quarter of an hour with an intracranial pressure above 70 millimetres of mercury, and the protrusion gradually increased until the proptosis was very marked (Figure VII). On dissection this was seen to be due to highly developed œdema of all the orbital tissues, most pronounced and earliest around the optic nerve, where it had the appearance of a crystal-clear jelly, and gradually diminishing to the fascia around the orbital muscles, beyond which it extended mainly forward as a very marked chemosis. Dripping from the nose commenced at about the same time as the eyes began to protrude, and continued at the rate of two drops per minute from each nostril so long as the pressure was maintained in the cranium. The fluid was clear, and showed only the minutest trace of protein content (acetic acid and boiling). After death of the animal the dripping continued at the same rate, and the proptosis continued to develop so long as the pressure was maintained. Each animal was carefully examined, and no sign of fracture in the ethmoid region was discovered in any of them. It is thus apparent that the mechanism of escape of fluid into the orbit and nose was one of pressure diffusion.

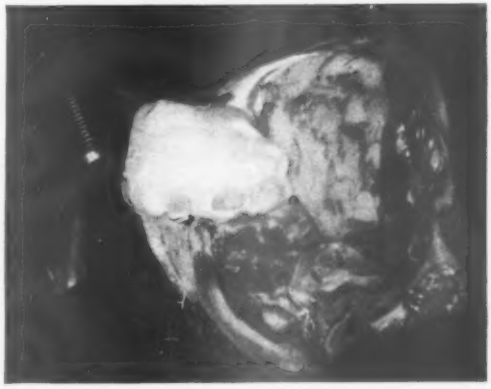

FIGURE VII. Dissection of orbit of dog after intracranial compression lasting fifty minutes (see Figure IV). The lateral wall and temporal muscle have been dissected away to show the œdema of the orbit extending from the optic foramen to surround and infiltrate the orbital muscles and encircle the cornea. The degree of protrusion of the eye may be judged by the cheek line.

When the sagittal sinus was opened along the whole extent of the cranial surface in recently dead animals, and the intracranial pressure was raised to 150 millimetres of mercury, a considerable flow of fluid appeared in it, as much as four drops per minute. This ceased

immediately on lowering the pressure. No point of appearance could be discerned.

DISCUSSION.

Intracranial Hydrodynamics.

The general physical principles of such a vascular system as here presented have been set out in the third section of this paper. From the experimental results it appears that the intracranial vasculature conforms to these principles in the essential phenomena. The junction of the arachnoid veins with the sagittal sinus corresponds to point of outflow of such a system, if the *dura mater* near the sinus is intact. The sagittal sinus in the intact animal is not made to collapse by raised intracranial pressure; it may be considered as a part of the extracranial venous system in this respect. The anatomical situation of this vein containing blood at a low pressure within the cranium and associated with arachnoidal villi may be of importance in the absorption of cerebro-spinal fluid. The general vasculature remains of approximately the same volume as before the pressure is raised—as little as one cubic centimetre of fluid may raise the intracranial pressure to the arterial blood pressure level. The reduction in rate of flow of blood is in direct agreement with the reduction of differential of pressure between point of inflow and outflow.

In one respect, however, the behaviour of the vasculature differs from that of the simple physical arrangement described above. In such a system, when the pressure surrounding such a tube system rises above the maximum available pressure, the collapse commences from the point of cuff constriction. In the cranium the veins remain full and collapse proceeds from the capillaries toward the arteries. The reason for such a divergence appears to be that when the intracranial pressure rises above the systolic blood level, the only avenue of escape of the blood from the veins is through the capillaries and arterioles toward the arteries, for there is no pressure greater than that at the compression cuff to force the blood toward the sinus. However, when the pressure rises above the maximum vascular pressure, there is the resistance of the small vessels between the veins and the arteries which collapse rapidly, so that the connexion between the veins and arteries is severed, a uniform pressure being left along the whole length of the vein; in these circumstances no movement of fluid will occur.

Medullary Reactions.

Of greater importance is the fact that, with a blood flow reduced in the proportion of 15 cubic centimetres per minute to three cubic centimetres per minute (Figure II), there was no reaction on the part of the cardio-vascular centre or the respiratory centre. Even when there was a reduction from 15 cubic centimetres per minute to five cubic centimetres in two and three-quarter minutes, there was a transient rise only of blood pressure, and some increase of the total volume of respiration. Only when the blood supply is cut right off from the

medullary vasomotor centre is there a rise of blood pressure. If the blood to the heart is oxygenated by artificial respiration, the centre remains capable of maintaining the mechanism for two minutes in the complete absence of blood flow, and apparently is subject to stimulation from afferent nerves in this period. Vagal effects are present, and, in the absence of both carotid sinus reflexes and artificial respiration, the rise of blood pressure is not so great as in the intact animal. This may be due to the absence of the reflexes from the sinus excited by the venous quality of the blood resulting from the apnœa. The rise of blood pressure is effected through the paravertebral sympathetic system from the medulla. That it was in any way due to excitation of the cells of the lateral horn of the spinal cord by anoxæmia was ruled out in the case of the recently cut cord by the absence of any blood pressure changes when the cord was compressed.

The apnœa produced is the result of paralysis of the respiratory centre. While apnœa lasts it is susceptible to stimulation neither directly nor reflexly. Apparently this centre may be mildly stimulated by the conditions associated with very reduced blood flow so long as there is some flow of blood (Figure II); but as soon as this meagre supply of blood ceases, then apnœa supervenes. Recovery is slow, and in the absence of external stimuli the general recovery shows a marked phase of slowed deep breaths. If, however, a strong stimulus from the carotid sinus is present when recovery occurs, then marked hyperpnœa may occur (Figure VI). The chemical condition of the blood at the end of a period of apnœa is only occasionally sufficient to excite this reaction.

These observations lay further stress on the observations that oxygen is necessary for the functioning of the respiratory centre; to lack of it the centre is not so much sensitive as vulnerable. They lay stress on the changing conception of cerebral anoxæmia also. Those remarkable reflexes of hyperpnœa and raised blood pressure which were ascribed to the direct reaction of the medullary centres to anoxæmia resulting from occlusion of the carotid and vertebral arteries, actually result from stimulation of nerve endings in the carotid sinus. The vasomotor and respiratory centres are not directly stimulated in this case. When the blood supply to the medulla is completely cut off the vasomotor centre excites a rise of blood pressure which is independent of these afferents, but remains sensitive to stimulation from them. The respiratory centre, however, "cuts out".

The observation of orbital œdema was made by Cushing and others. It is the experimental basis of the present conception of papillœdema in cases of prolonged rise of intracranial pressure. If this perilobar (perineural) permeation of fluid is conceded, then the occurrence of rhinorrhœa in the high experimental pressures may be explained on a similar basis (Speransky[21]).

CONCLUSIONS.

1. During elevation of intracranial pressure the sagittal sinus does not collapse; the intradural vessels remain of normal or increased (Forbes) diameter until the intracranial pressure exceeds systolic arterial blood pressure. The veins then remain full, but the small vessels and arteries collapse.

2. Blood pressure and pulse rate do not show any alteration until intracranial pressure is at the level of systolic blood pressure (Cushing), when a rising blood pressure and reduced pulse rate are brought about through normal physiological channels.

3. The respiratory centre shows slight sensitivity to greatly reduced blood flow, but in the absence of blood flow is insensitive to stimulation.

ACKNOWLEDGEMENTS.

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THE SURGICAL TREATMENT OF DETACHMENT OF THE RETINA.[1]

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THE surgical treatment of detachment of the retina must surely rank as one of the greatest advances in ophthalmic surgery which this century has seen. We all know the utter hopelessness with which we used to advise a patient suffering from this dread disease to go to bed for a month or six weeks, and how in 99% of cases we found not the slightest improvement in the condition after that long weary confinement. By surgical means we are now able to achieve a complete cure of the condition in something like 45% to 50% of the cases; and it is my own feeling that if the cases are selected, the percentage of complete successes amounts to something like 75.

In this short paper I do not propose to go into the history of the surgical treatment of detachment of the retina; that can easily be found in many of the excellent papers and books which have appeared on the subject from time to time. Rather, I hope to give you some personal experiences in the surgical treatment of detachment.

In Wellington I think I have tried every known method except electrolysis, and I am hoping to use electrolysis in one case in the very near future.

I propose to discuss my subject under the following headings: (i) some factors influencing success of operation; (ii) choice of operation, during which I shall give you some of my experiences with the various types of operation; (iii) after-treatment; (iv) end results.

I am not going to give you a series of statistics, as so far I have not treated a sufficient number of patients to warrant my drawing any statistical conclusions from them.

SOME FACTORS INFLUENCING SUCCESS OF OPERATION.

I have stated previously that if the cases are selected it is possible to achieve almost 75% of cures. Let us see which cases are likely to be successful.

Age.—On the whole, the age of the patient has very little to do with estimating the chances of success or failure. It is my impression that in the young adult treatment is a little more successful than in the elderly patient, but that may be because a greater percentage of

[1] Read at the annual meeting of the Royal Australasian College of Surgeons, Auckland, January, 1937.

my patients have been young adults. I assisted a year ago in the treatment of a patient well over seventy, and here the result was just as good as in a younger patient. Of course, in young children who will not stand local anæsthesia, the chances of obtaining a successful result are much less.

Last year I operated twice on a very nervous child, aged ten years, who had a very extensive inferior detachment. I was compelled to give her chloroform, and I am sure that the vomiting had something to do with the first unsuccessful operation. The second operation was a complete success.

In the literature available to me I can find no record of a successful result in a patient as young as this one.

Duration of Detachment.—It has been my experience that the sooner a patient with a detachment is operated on, the better the chance of a successful result. All observers in England and on the Continent are in agreement with this statement, but do not let it prevent you from attempting treatment of a detachment which has been present for some considerable time. One of the most successful operations that I have performed was in a youth of eighteen years in whom the detachment had been present for at least two and a half years.

Extent of Detachment.—The extent of the detachment has a large effect upon the ultimate result of the operation. The greater the detachment, the less the chance of a successful issue. The largest detachment that I have successfully replaced was one which involved a little more than half of the retina. I have operated in cases which involved almost the whole of the retina. In certain cases I have performed as many as three operations on the one eye. But in no case did I obtain any improvement at all, so that now I would not operate on any patient whose retina was more than two-thirds detached.

Position of the Detachment.—It has been my experience, and also that of my colleagues in Wellington, that detachments in the inferior part of the retina do much better than those in the superior part of the retina. I have not seen attention drawn to this point in any reports elsewhere, but it has been a very important point in all my cases, so much so, that I always operate on superior detachments with much trepidation as to the ultimate result. I feel that the position of the patient following operation favours reattachment in inferior detachments. I have thought of raising the foot of the bed in the treatment of the next superior detachment, but I doubt whether the patient would tolerate the position for any length of time.

The Area of Pigment.—I have found that detachments which are limited by an area of pigment—what really looks like the line of a chorioiditis—seem to have a particularly good prognosis. It is my feeling that such detachments are really caused by some form of chorioiditis which causes an effusion between the retina and chorioid, and thus brings about detachment of the retina. The upper edge of such a detachment is delimited by a pigmented border. All the patients in whom I have seen this condition have been young subjects, and they have all had excellent results following operation.

Holes in the Retina.—I have not as yet operated on a sufficient number of patients to be able to give any very definite opinion on the effect of the position, size, shape and number of holes in regard to the ultimate prognosis following operation. Harrison Butler states that he is unable to find a hole in as many as 50% of his cases. I am afraid I cannot agree with that statement, as I can find a hole in at least 90% of my own cases. If the patient is put to bed for a few days an undiscovered hole will frequently manifest itself. It has, however, been my experience that in the cases treated by diathermy it is not nearly so necessary to mark out accurately the position of the hole as one is led to believe from reading the reports in the literature from abroad. I may be quite wrong in my deductions on that point, but I can honestly claim that my results to date are just as good as those claimed in England; and I must confess that I do not go to a great deal of trouble to mark the hole. Do not let me mislead you into thinking that I disregard the hole altogether, but I feel that I can mark it out nearly enough by just using an ophthalmoscope. I should like to hear the experience of some of those present on this point. I examine the eye very carefully the night before operation, and by means of the ophthalmoscope fix in my mind the position of the hole. I do the same again almost an hour before operation. In two of the most successful cases that I have had I was unable to find any hole at all. I feel that the very large ragged hole offers the worst case for operation; but, as I said previously, my conclusions on the effects of holes on the ultimate prognosis are at the moment, because of the insufficient material to date, very much in the melting pot.

Involvement of the Macula.—Involvement of the macula has no effect whatsoever upon the ease or otherwise of reattachment of the retina by operation. When the macula is involved, it does, however, have a very great effect upon the prognosis so far as ultimate central vision is concerned. In such a case, even though the retina is completely reattached, the central vision may show little, if any, improvement at all. Some observers have stated that if the macula is detached for more than five or six days, the central vision will not improve much following operation. In one of my own cases the macula was involved most certainly for at least a month, and following operation the central vision improved from $^{6}/_{36}$ to $^{6}/_{9}$. In two other successful cases with macula involvement the central vision remained the same following operation as before, namely, $^{6}/_{24}$. When the macula is involved I am, therefore, very guarded in my prognosis about central vision.

CHOICE OF OPERATION.

At the present moment it is my practice in all operations to use Safar's technique. In the last five cases in which operation was performed by this technique I have had 100% successes, but of course they have all been picked cases. In six cases I used Gonin's technique, and I must confess that in none of them did I obtain any improvement at

all. I used an elaborate technique to mark out the position of the hole or holes, and carried out Gonin's instructions, but in no instance could I satisfy myself that there was any permanent improvement. This is probably a terrible confession, but it is the truth. I found the operation difficult, and it is possible that some minor detail in my technique was wrong, as Gonin himself undoubtedly obtained results. But I have noticed recently that some other observers in England have stated that they also found it difficult to obtain results from Gonin's method.

In some of my cases the condition was very bad, and in at least three of them I would now regard failure as almost certain to occur by any technique. These cases partly account for my bad results with Gonin's method. Nevertheless, I would not attempt now to treat any detachment of the retina by Gonin's method.

I have tried multiple trephining, but it is not nearly so easy as the operations involving the use of the diathermy. So far it has been my experience that diathermy operations (Larsson's and Safar's) are much the easiest to perform, and they give me the best results.

In an earlier part of this paper I stated that so far I had not tried the method of electrolysis. Theoretically the electrolysis method appears the best.

I will not weary you by a complete description of Safar's technique, with the details of which I am sure you are conversant. (I have described the operation in detail in *The New Zealand Medical Journal,* February, 1937.) Rather do I wish to lay stress on what appear to me to be some of the important points in the execution of the operation.

1. Repeated ophthalmoscopic examinations must be carried out in order that the surgeon may become thoroughly familiar with the position of the detachment and the hole or holes. The known position of the various recti muscles will help to fix the position of the hole in mind, and it is moderately easy to fix its position further in disk diameters from the *ora serrata.*

2. Always see that you have good exposure of the area to be operated on. Plan your incision so that you will be operating over the area of greatest detachment, provided of course that you can also include the region of the hole. I never hesitate to cut partly through one or two of the recti muscles if by so doing I am enabled to obtain better access. By placing traction sutures in the conjunctiva between the limbus and the edge of the incision it is possible to pull the eye well up, and thus to increase greatly the area of operation. I also use two small vulcanite retractors which were made in Wellington and which fit into the lower edge of the wound. Besides acting as very efficient retractors, they prevent sparking between the diathermy points and the eyelids.

3. The use of a pair of dividers, as used in school, *plus* a small metal rule, is a very easy method of ensuring that at no time will your diathermy points approach nearer than 9 millimetres to the limbus. They also help to keep the position of the *venæ vorticosæ* continually

in mind. Both these instruments can be boiled and sterilized with the rest of the instruments.

4. The ideal of Safar's operation is to encircle the region of the maximum detachment and hole with a series of diathermy points. This is usually done by means of about four multipointed electrodes. The ideal is to get all the electrodes in position and not to remove any until the last is in place. By doing the operation in this way a hard eye is ensured. I must confess that I have never been able to get four multipointed electrodes into the eye at the same time. At the most, I get two to stay in, and I have to finish up with a single-pointed electrode: but with a single-pointed electrode I do not find a soft eye to be a very great inconvenience.

5. In using Safar's electrodes the surgeon must always remember to keep slight pressure on the electrode as he switches on the current. I have always found it a great temptation to release the pressure on the electrode as soon as I switch on the current. The patient also quite often flinches at the same time and pulls his head away, with the result that instead of the eyeball being penetrated a nasty little burn is caused on the sclera. If you always remember to keep pressure on the electrode this occurrence can be readily avoided.

6. Before the electrodes are applied to the sclera it is most essential to have the sclera quite dry and clean. This can easily be done by a little careful dissection. Firm pressure with a swab stick and with a few drops of adrenaline usually controls all hæmorrhage. It is most important not to commence application of the electrodes until all bleeding has been stopped.

AFTER-TREATMENT.

To ensure a successful result it is most important that the after-treatment should be faithfully carried out. The essence of the after-treatment can be summed up in absolute rest of the eye that has been operated on.

Both eyes must be firmly bandaged, and it is my practice to keep them bandaged for at least two weeks. If I obtain a successful result I may keep them bandaged for as long as three weeks. The patient should be nursed in that position which ensures the maximum amount of drainage of the subretinal fluid, and at the same time allows the detachment to be in the most dependent part of the eye. Thus a patient with an inferior detachment should be nursed in Fowler's position, although I must confess that I have found a strictly recumbent position gives equally good results in an inferior detachment. A patient with a superior detachment should have the foot of the bed raised, while a patient with a temporal detachment should lie on the same side of the face. It is most essential to instruct the nursing staff in the need for absolute rest.

I do not look at the fundus until the sixth day. I have formed the opinion from my own cases that if the retina is not completely back in place on the sixth day, no further rest will reattach it. I know that

observers in England have reported cases in which the retina has not become completely reattached until two or three weeks after operation, but that has not been my experience at all.

When the recumbent position is used I give the patient a pillow at the beginning of the third week, and gradually increase the height of the pillow until at the beginning of the fourth week he is sitting up. Towards the end of the fourth week he is allowed out of bed. The patient must be instructed that no heavy lifting or strenuous exercise is to be undertaken until six months after operation.

I realize that the after-treatment is somewhat drastic, but I have had very little trouble in persuading my patients to submit to it.

END RESULTS.

There is no doubt about the fact that it is possible by surgical means to replace a large percentage of detached retinæ.

In most cases after the retina has been replaced it takes some time for it to recover its full power. This is shown by the fact that, although ophthalmoscopically the retina is completely replaced, you may be able to obtain a full field with only a 5 millimetre test object. It may take some months before you can obtain a full field with a 1·5 millimetre test object, and in certain cases you may never obtain a full field with a test object as small as 1·5 millimetres. It seems as if the retina during its detachment loses some of its power of finer differentiation. On the other hand, I have had cases in which immediately after reattachment it was possible to get a full field with a 1·5 millimetre test object.

I have stated previously that in a great number of cases in which the macula is detached not much improvement takes place in central vision following reattachment. Most cases of detachment of the retina progress until the retina becomes almost completely detached, so even though a reattached retina does not completely return to its normal functioning power, operation at least prevents the rest of the normally functioning retina, including in a great number of cases the macula, from becoming detached too. However, most reattached retinæ do regain a very considerable amount of their normal functioning, and once again form a very useful part of the visual apparatus.

There is no doubt that diathermy and the other methods of replacing the retina cause a fair degree of trauma to the retina. This is shown by the fact that the spots at which the diathermy points have penetrated the eye are very easily seen with an ophthalmoscope, and in most cases they are of fair extent.

One of our ideals should be to reduce this trauma as much as possible. I am not quite sure how it may be done—possibly the new method of electrolysis will cause less trauma.

Still, despite some disadvantages, there is no doubt in my mind that the surgical treatment of detachment of the retina is a very useful operation, and that it will remain as the best means of treatment of this dread disease.

HERNIA AND ITS OPERATIVE TREATMENT.[1]

By Balcombe Quick,
Melbourne.

Since hernia has been an infliction of mankind from the beginning of time, it is small wonder that the subject engaged the attention of the earliest physicians. Celsus described the application of a truss, a bandage with a cushion pad of rags "to repel the intestine", and even described an operation which he himself, however, had not the temerity to perform. He set his face, curiously enough, against any operative interference in strangulation.

Before the earliest dawn of surgery, in the second century A.D., Galen was teaching that inguinal hernia was due either to a gradual distension of the *processus vaginalis* or, in the event of the sudden descent of a large hernia, to actual rupture of the processus and of the aponeurotic structures which lie over it. He had no opportunities for dissecting the human body, no pathology of the living to guide him; his deductions were drawn from the study of animal tissues alone. In the sixteenth century, as the result of his dissections of human bodies, Vesallus was able to expose the errors into which Galen had fallen; but so great was the authority of Galen's name that for another two hundred years his erroneous teaching was still current.

In his "Treatise on Ruptures", published in 1775, Percival Pott, after shrewdly remarking that "the generality of mankind look upon a rupture as an imperfection in their form, as a disease which impairs their strength and lessens the generative faculty", continues thus:

> I intentionally avoid saying anything about the old doctrine of the difference between dilatation and laceration of the peritoneum, it being now generally known and acknowledged that to whatever size the hernial sac may extend, and however large its contents may be, it is merely dilated and hardly ever burst or broken.

Thus Pott, in finally disposing of this fallacy, ended one of the many controversies in which the subject has been continually involved.

One hundred and twenty-five years later, in 1899, Hamilton Russell first enunciated his theory of the congenital origin of the oblique hernial sac and thus began the demolition of yet another fallacy. I take it that I may paraphrase Percival Pott and assume without possibility of controversy that "to whatever size the hernial sac may extend, and however large its contents may be", that sac is of congenital origin. This state-

[1] A lecture delivered at the Royal Australasian College of Surgeons, Melbourne, on June 30, 1936.

ment is, I think, true of all inguinal herniæ except the direct variety, the majority of which appear to be acquired.

The controversy which appeared for so long to be inseparable from the subject of the origin of hernia is still equally in evidence as far as its treatment is concerned. New operations, or rather, variations of old operations, appear with unfailing regularity. There seems to be something strangely attractive to certain surgical minds in the various permutations and operative combinations into which a sac, an aponeurosis or two, and a conjoined tendon may be built up.

If some sort of apologia is necessary for my choice of subject this evening, I can at least claim that I have no new combination of tissues to submit to your consideration, and I have no more to advocate than a procedure designed to restore normality to the inguinal canal.

The term hernia, unqualified in any way, conveys to all minds the common oblique inguinal hernia which particularly affects the male in his youth, his middle and his old age. The extreme prevalence of the condition and its effect upon human industry and happiness confer upon the subject an importance greater than that usually ascribed to it, and a stocktaking from time to time is not out of place.

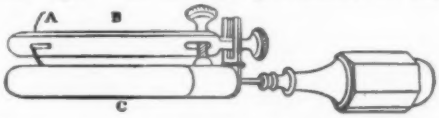
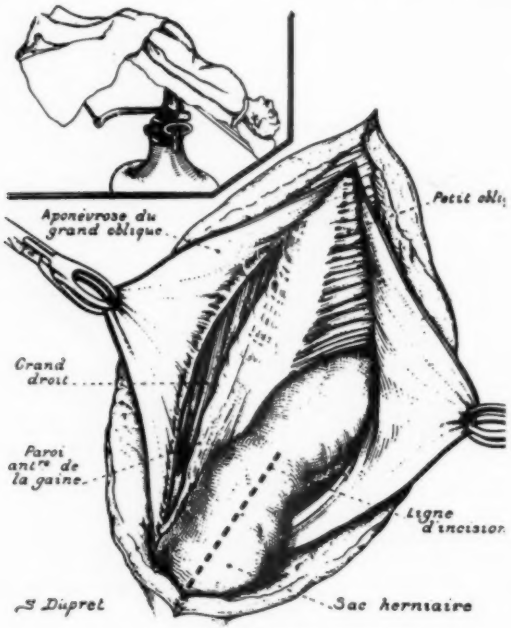


FIGURE I. The instrument of Leroy or Wutzer. After reduction of the hernial contents the rod (C) was made to invaginate the scrotal tissues and pass up into the canal. The curved needle (A) was then caused to emerge from the rod and penetrate the tissues overlying the upper end of the canal. The ivory plate (B) was then pressed down upon the skin over the canal and fastened there in the manner indicated.

It has been estimated that one out of every sixteen persons born will sooner or later acquire a hernia, and that every year some 2,000 deaths in Great Britain are due to strangulation of a hernia. This figure is for recent years; I am not aware of any to show what the mortality may have been in the days before operation freed the great majority from that risk.

How comparatively recent those days are is surprising. The name of Spencer Wells is not so very remote, and yet it was he who in 1854 introduced into English surgery the fearsome apparatus of Leroy or Wutzer (Figure I). The ninth edition of "Erichsen's Surgery", published in 1888, speaks of its use as being moderately safe and followed by not a few successes. In those pre-Listerian days the danger of peritonitis was ever present, and it was not until after the birth of modern surgery in 1867—Lister's year—that the idea of open operation for hernia began to be generally entertained. Between 1880 and 1890 Banks, Barker, McEwen and Czerny devised their procedures, but all suffered from the defect of imperfect removal of the sac, for in none was the canal opened up. It is probable that only patients with the largest and most crippling herniæ were submitted to operation, and that in these persons the canal had become so straightened out that the need for a higher approach was not apparent. The aim in all these

operations seems to have been to close the external ring after such removal of the sac as was possible. That reliable authority Jacobson (third edition, 1897) is rather guarded in his attitude at that period. He discusses the justifiability both of the operation and of the use of the term "radical cure", and the advisability of wearing a truss after operation. When a truss would retain a hernia, he did not advise



LARGE SCROTAL HERNIA. RADICAL CURE.

FIGURE II. Taken from Victor Pauchet's "Practical Surgery", showing the use of the Trendelenburg position.

operation at all. The influence of pre-Listerian days is still apparent in that the chapter on strangulation has pride of place.

The year 1890 stands out in the history of the evolution of operation for hernia, for it saw the publication of the methods of Bassini and Halstead with their opening of the inguinal canal. The immediate effect was remarkable, as witness that upon procedure at St. Bartholomew's Hospital.

Table (from "St. Bartholomew's Hospital Reports").

In 1887 5 operations for radical cure of inguinal hernia.
In 1897 113 operations for radical cure of inguinal hernia.
In 1907 276 operations for radical cure of inguinal hernia.

NEW TREATMENTS AND OLD.

When one reads the history of hernia and of the attempts that have been made through the ages to effect its cure, it seems doubtful if any really new method in treatment, beyond minor variations in details of operative technique, is ever likely to arise. Had I been addressing you some twenty-five years ago I might have been bringing to your notice the

FIGURE III. Operation for hernia, Roland, thirteenth century. Taken from *La presse médicale*, 1902, Volume x, page 603.

results of silver wire filigree implantation by the method of Bartlett and McGavin, which about that time enjoyed a considerable popularity for a number of years. You might or might not have been aware that in the middle sixteenth century Ambroise Paré was using a golden wire with some success in the treatment of the sac. McArthur and Gallie, the originators of the *fascia lata* graft, were antedated by decades by the

enthusiast who proposed the transplantation of periosteum for the same purpose. Those who have employed the Trendelenburg position to secure the return of the contents of a difficult hernia (Figure II, inset) were simply following a method advocated and figured by Roland in the thirteenth century (Figure III). Within the last few years it appears that an injection treatment, tedious and uncertain though it must be, has become popular in certain quarters. Precisely such a treatment was in use one hundred years ago, except that red wine, galls, iodine and even blood constituted the injection fluid in place of the synthetic sclerosing combinations of today. There is no significance attaching to this vogue; it is merely evidence of the very general desire to avoid a cutting operation; but on the other hand the significance of the multitude of operations which have been and still are being devised for the cure of hernia is very real. In fact, there is a double significance. There is an unfailing implication in the first place that no operative procedure can be relied upon with certainty to bring about cure, and, secondly, that there has been a failure to grasp the principles which should govern the treatment of hernia.

I shall examine in turn the facts upon which these statements are based.

RECURRENCE RATE AFTER OPERATION.

First, the success or failure of any operation for the cure of hernia is obviously determined by the recurrence rate, and though the discrepancies which appear statistically in this respect are very great, it is apparent that a goodly number of failures must be debited to even the most experienced surgeons. It is not unlikely, then, that unpublished figures and the results of the occasional operator may show a still larger proportion of failures. All statistics are open to the usual charge and due allowance must be made for two factors at least: the number of children in the series and the time after which the follow-up was conducted. Follow-ups seem extraordinarily difficult to conduct in this class of case, and, contrary to general belief, the risk of recurrence is by no means confined to the first year after operation.

Coley's figures, for example, from the Hospital for Ruptured and Crippled, New York, show a general recurrence rate of 6·8%, but if children are excluded (some 1,000 in number, and in none of whom was recurrence found to have occurred) and all other than the adult male patients with oblique hernia, the rate rises to 8·7%; but less than one-third of all the patients could be traced. A Bassini type of operation was the rule. Surgeons of other representative American clinics admit from 8% to 11% of failures.

Max Page recently reported a recurrence rate of 11·5% among the London police. On the other hand, large series—10,000 or more cases from German and Russian clinics—show a recurrence rate in the region of 5% (Bassini and Roux operations); and from Guy's Hospital a recent series of adult patients with hernia showed 6% of recurrences. These

figures should, as Wyllys Andrews says, "surely shock surgeons out of their serene confidence that we are doing justice to these patients".

So much for the first implication. The second implication from this multiplicity of methods is that there has been a failure to grasp the underlying aim or principle of treatment—restoration of the inguinal canal to normal.

DEVIATION FROM NORMALITY IN HERNIA.

The deviation from normality which constitutes a hernia consists of (*a*) the presence of a sac and (*b*) an aperture of increased but variable size in the floor of the canal at its lateral end, that is, at the internal abdominal ring. Still a third deviation from the normal, but one which is less generally recognized or admitted as a cause of hernia, is some defect in the level of origin or of the insertion, or both, of the internal oblique muscle, the internal and external rings respectively being thus left unguarded. Disregarding for a moment this third possible deviation or factor in the hernial state, it may be said that normality will be secured by removal of the sac and by restoration of the internal ring to its former size and position. This will once more bring about such a relationship between the ring and the curved lower border of the internal oblique that this muscle covers the ring during effort.

If the general principle is accepted that the sac constitutes the primary cause of the hernia, the first aim of treatment should obviously be complete removal of the sac, and this is undoubtedly an integral part of all operations practised at the present time. Such is the aim at all events. In different hands a greater or a lesser degree of success may be attained.

The second deviation from normality is an aperture of variable size in the floor of the canal at its commencement at the internal abdominal ring. The normal ring is snugly filled by the spermatic cord, but the presence of a hernia or even of a potential hernial sac must imply an enlargement of the ring, which is simply an opening in the general fascial lining of the abdomen known in this region as the transversalis fascia, and which is continued over the cord as the infundibuliform fascia.

The Enlarged Internal Ring.

Such an enlargement of the internal ring tends in general to be progressive. The increase is almost entirely in a medial direction, so that in large herniæ the obliquity of the canal is compromised or lost, and the internal becomes more or less subjacent to the external ring.

In the lesser degrees of such enlargement one finds that the lower margin of the internal ring is beginning to emerge from under the protecting cover of the edge of the internal oblique. To fulfil the second part of treatment then it will be necessary to restore the size of the internal ring to normal, and in so doing to place it once more under the protective cover of the internal oblique muscle.

Now there are obviously two ways of altering the relationship of the enlarged internal ring to this protective lower border. The first is by closing the lower portion of the dilated ring so that the cord passes through the upper and protected portion, and the other is by suturing the origin of the internal oblique muscle down to a lower level of Poupart's ligament as in Bassini's operation. The former of these methods alone will restore the canal to normality, and is therefore the one to be employed.

Mention was made of a possible third deviation from normality—such a defect in the structure of the internal oblique muscle that it was precluded from exercising the function of a half sphincter. This may, I believe, be summarily dismissed. If it is true, it is impossible to conceive how any hernia in any child, or any adult for that matter, can be cured by removal of the sac alone. Dismissing this factor from consideration, therefore, we may say that, given complete removal of the sac, successful reconstruction of the internal ring and floor of the canal and primary wound healing, cure of the hernia should result.

THE TRUE BASSINI TECHNIQUE.

It was the appreciation of the fact that in established hernia the internal ring was enlarged, and that the floor of the canal was thus weakened and its obliquity compromised, that induced Bassini, the pioneer of modern methods of so-called repair of the canal, to attempt to strengthen the floor. This he did by suturing not only the lower muscular border of the internal oblique and, more medially, the conjoined tendon to Poupart's ligament in the well-known fashion, but also to include in his sutures the *fascia transversalis*. Thus Bassini endeavoured to combine both stratagems in his method. Wyllys Andrews, who visited his clinic at Padua in 1899, has made this abundantly clear by writing his description of Bassini's technique as follows:

> Now the dissection of the peritoneum away from the internal ring has also had the effect of loosening the transversalis fascia and internal oblique muscle. . . . . These as everyone knows are the structures to be included in the Bassini operation in the first or deep line of sutures intended to repair the enlarged ring to its normal size.

Other surgeons, such as McEwen, planned their operations with this end in view, and the older surgeon-anatomists—Scarpa, Cooper and others—showed clearly in their dissections of hernia that the deficiencies of this fascial layer play a very definite part in the pathology of hernia. Either the ring might be enlarged or the portion next to Poupart's ligament might be thinned out and stretched.

It is unnecessary to emphasize the fact that the great majority of surgeons who carry out what they term a Bassini operation for oblique inguinal hernia concern themselves not at all with, nor give thought to, the transversalis fascia and the size of the abdominal ring. Although the congenital origin of the sac and its paramount importance are now almost universally accepted, particularly in this country perhaps, it is a

curious commentary on human nature that Bassini's operation is so often performed by those who claim to subscribe to the saccular theory. One is reminded of the old lady who always crossed herself when the devil's name was mentioned, maintaining that politeness cost nothing and that it was just as well to err on the safe side. Such, no doubt, is the subconscious argument of many surgeons in the matter of operation for hernia, and so it comes about that, although in full agreement with all Russell's teaching, they carry out the operation somewhat on the Bassini lines, but without any repair to the transversalis fascia. There has been an error of omission. There has been added to this, moreover, an error of commission.

The Internal Oblique Sphincter.

The function of the internal oblique muscle here is that of a half sphincter. Just in so far as any effort or straining which involves the use of the abdominal muscles raises the intraabdominal pressure, so, *pari passu*, the curved lower border of the internal oblique muscle straightens, shortens, approaches Poupart's ligament, and denies any escape to the abdominal contents through the region of the internal ring.

To those who have operated for recurrent herniæ there is no need to describe the results of a Bassini suture—the muscle is no longer found tidily fastened down to Poupart's ligament. In its place is a rather confused layer of thin scar tissue which represents the portion of the muscular border that has degenerated as a joint result of tension and anæmic necrosis. It has been shown, moreover, by the experiments of Seelig and Chouke that red muscle and fibrous tissue will not unite following any method of suturing, save in the presence of infection. So that not only has this method of repair of Bassini's failed to produce the desired continuous and permanent muscular barrier to any attempted exit through the internal ring, but it has destroyed that very portion of the internal oblique muscle which was designed to defend the approaches to the canal at the appropriate moment. It appears, then, that of the two methods of adjusting the relationship of the internal ring to the protective portion of the internal oblique muscle, Bassini's method is inferior to a simple closure of the lower portion of the fascial opening or defect in such a way that the cord again emerges under cover of the muscular fibres.

That other deviation from normal structure which allegedly operates in the production of a hernia—such a high origin of the internal oblique muscle or such a low insertion of the conjoined tendon that normal sphincteric action is impossible—would render more impracticable than ever the restoration of a muscular barrier by any suturing methods involving this muscle, and the canal mechanism must therefore, in such an event, depend for its integrity upon a strong fascial floor alone.

In what way do the operations as commonly practised fall away from the theoretically correct principles?

THE RUSSELL OPERATION.

I shall consider first simple removal of the sac as practised by adherents of the saccular theory.

Russell was insistent upon high removal of the sac, and he drew a very clear distinction between the method to be employed in the cure of a small hernia and that for a large hernia with much damage to the posterior wall of the canal. In recent and favourable cases the fascia was to be pushed aside and the ligature applied to the peritoneal sac alone. He claimed that after the stump had been allowed to drop back, it would lie within the fascial layer, the opening in which would close over it, and that a mere scar in the fascia would result. Indeed, in young children he was accustomed to content himself simply with torsion of the sac and did not apply a ligature to it. In large herniæ of long standing the sac and its overlying fascia were twisted up together. He described this as "closure of the hernial opening by means of the peritoneo-fascial layer of the abdominal wall". This had the additional effect of approximating to some extent the internal oblique and transversalis muscles to Poupart's ligament. I believe that most of those who follow Russell's method practise separation of the peritoneal sac from its overlying fascial coverings until its neck is revealed, and that after torsion, ligature and division, the stump is dropped back and the resultant gap in the fascial layer is left to care for itself. I question very much if such an opening will always "close over it [the stump] and a mere scar result". I am sure that to apply such a method to a large hernia is to invite recurrence. The occasional recurrences at Russell's hands were, I am convinced, due to neglect of the aperture in the fascia in the herniæ of moderate size. That he had some suspicion of this sort occurred to me on one occasion when I operated upon such a case in his presence. I was taken to task for carrying the isolation of the sac so far as being quite unnecessary and undesirable. It was only after long thought that I concluded that it was not the high separation of the sac of which he disapproved, but the consequent enlargement of the fascial opening. A defect or weakening, even if temporary, as he believed, of the floor of the canal must be produced thereby. To that extent the Russell operation as practised upon the smaller herniæ falls away from the theoretically correct principle. Such a falling away is less apparent in the case of large herniæ in which he twisted up and removed the combined peritoneo-fascial layers together, but still the procedure is not quite ideal.

Of all such other commonly practised operations as include the suturing of the internal oblique muscle and conjoined tendon to Poupart's ligament the criticism just made applies—that further damage to the cause inevitably follows. Towards the inner end, where the conjoined tendon is sutured to Poupart's ligament, a laceration and tearing apart of the tendinous fibres are inevitable, if the sutures hold. At the outer end scar tissue replaces the lower muscle fibres. Russell claimed, too, that those who practise muscle suture in Bassini fashion fail to acquire

the art of high removal of the sac, and that the repair then carried out is more one of surgical deficiencies than of anatomical defect.

A study of recurrent herniæ is not without interest. Following operation of the Bassini type, about four out of seven recurrences are at the medial end of the canal; that is to say, they are direct. Such a hernia is not a recurrence in the true sense; it is a post-operative ventral hernia due to damage inflicted upon the conjoined tendon when fastening it down to Poupart's ligament. The fibres of the tendon pull apart and the recurrent sac is often found to be passing through quite a narrow gap in the tendon with well defined margins.

I do not think I have ever seen, nor would I expect to see, such a hernia follow simple removal of the sac. When recurrence then occurs it is, I think, always at the outer end, and is due either to inefficient removal of the sac—an unlikely event—or to failure to repair properly the enlarged internal ring.

I have already indicated what I conceive to be the defect of the Bassini operation as commonly practised. The same criticism applies to all those other methods, such as those of Halstead, Schmieden, and Ferguson, in which the fibres of the internal oblique muscle are sutured to Poupart's ligament, whether in front of or behind the cord. In Ferguson's operation, be it noted, "a few stitches are applied to narrow the internal ring" before the internus is drawn down in front of the cord.

THE ANDREWS-GIRARD OPERATION.

The operation of Wyllys Andrews, or Girard, as it is known on the Continent, is in a different category. In this imbrication operation the first step is a repair of the enlarged or weakened internal ring and old floor of the canal by suture of the transversalis fascia in its higher and stronger part to Poupart's ligament, preserving its normal relationship to the cord. The next step is the suturing down of the upper leaf of the external oblique aponeurosis in the same fashion and still behind the cord, this strong fascial layer being thus used to form a new floor to the canal. The operation is completed by turning up the lower leaf and fixing it by a line of sutures in front of the cord. The outstanding feature of this operation is that the muscular fibres of the internal oblique remain inviolate, and thus Andrews describes it as a repair of the inguinal canal by white fibrous tissue alone.

My criticism of this operation is that it has a distinct field of usefulness when weakening of the transversalis fascia is very marked and when no preparations have been made for the use of *fascia lata* in the repair of the floor of the canal. The restoration that it brings about is by no means anatomical, however, and my belief is that throughout the whole of surgery form and function are so intimately related that anatomical restoration should be an ideal to be sought.

You have listened more or less patiently to a great deal of theorizing. As Pitzman has said, however, "if the theory of the operation is better, *ipso facto*, the practical results will be better".

Let me now speak of some cases which may lend substance to theory.

I had occasion some years ago to operate upon a man who nine or ten hours earlier had burst his small intestine by leaning forward upon an ill-fitting truss. An early peritonitis was present, but it seemed that no harm could result from passing a forceps down the sac from within, seizing the fundus of the sac, which was about two inches long, drawing it into the abdominal cavity and tying it off at the neck. It was then sutured down securely to the parietal peritoneum. The sac was thus removed from the canal with absolutely no interference with the structures superficial to it—an experiment I had long wished to carry out. That hernia had recurred within three months. A subsequent operation with repair of the enlarged internal ring has now been successful for upwards of three years.

Next take the case of a boy of three upon whom I assisted Mr. Hamilton Russell to operate, following his usual technique. All went well for fourteen years, when, following an operation for appendicitis, the hernia recurred. I am not aware what operation was then carried out, but it was successful.

FIGURE IV. Right scrotal hernia descending below mid-thigh.

I translate this story as the uncovering of an enlarged internal ring following the weakening of the lower fibres of the internal oblique by a McBurney incision.

A year ago I operated upon a colleague for a recurrent hernia by the method of Gallie. Some twenty years ago he had had a double hernia operated upon by Hamilton Russell and myself. My senior took the larger hernia and entrusted me with the smaller sac. My technique was a faithful imitation of his own. With the passage of years and time's theft of muscle tone, the weakness of the larger defect in the transversalis fascia was revealed.

Finally, let me show you the largest hernia that Russell himself had operated upon at that time, at which operation I was privileged to assist him (Figure IV).

In this case the peritoneo-fascial layer, not the sac alone, was twisted and transfixed, and thus the defect in the transversalis fascia was to a considerable degree rectified. I had this patient under observation for many years afterwards, and though a hydrocele followed there was no recurrence of the hernia in the true sense, only a slight weakness of the whole inguinal region remaining. I cannot conceive of a more successful result by any method.

THE AUTHOR'S OPERATION.

The operation that I perform has little to distinguish it from those of Connell (Figure V), Pitzman, and Seelig, all of whom aim at removal of the sac, repair of the internal ring and nothing else. Though their methods differ slightly in detail, the underlying principle is the same.

The method is shortly described as follows. After exposure of the spermatic cord in the inguinal canal, the sac is identified and uncovered by pushing aside the overlying cremasteric fascia at a convenient site, usually, in the case of a small hernia, an inch or so from the internal ring (Figure VI).

The delicate infundibuliform (transversalis) fascia, lying between the cremasteric fascia and the sac itself, is here rather imperfectly

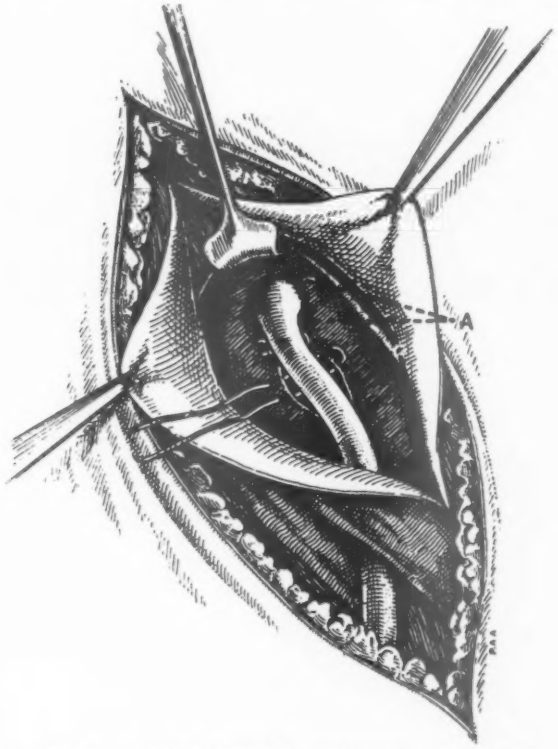


FIGURE V. Repair of internal ring (A) in oblique inguinal hernia by the method of F. G. Connell. (Redrawn from *Surgery, Gynecology and Obstetrics.*)

differentiated from the cremasteric fascia, but as the internal ring is approached the infundibuliform fascia becomes a more and more definite entity. As this fascia is stripped from the sac the assistant grasps successive segments of its circumference with artery forceps. With care, a complete fascial tube, through which the sac passes, is finally demonstrated in this way, held open by those forceps which have been applied

FIGURE VI. The hernial sac, still covered by the infundibuliform fascia. The dressing forceps, directed towards the fundus, is starting the separation of the fascia from the sac and of the sac from the cord.

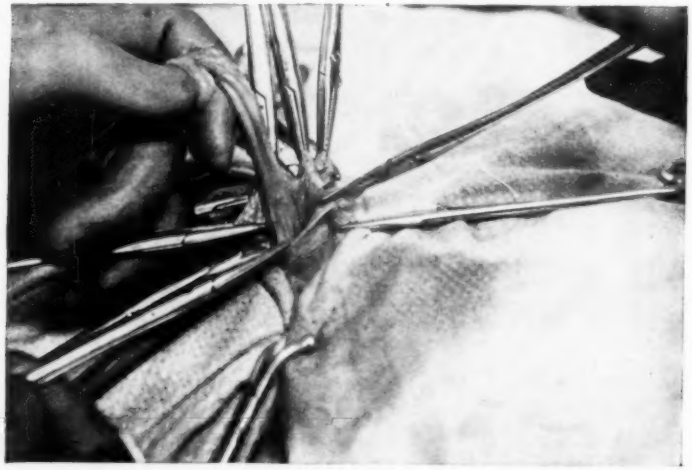


FIGURE VII. The sac, held in the left hand, is seen passing up through a fascial tube to the margins of which a number of forceps are applied.

at or just above the site of the internal abdominal ring (Figure VII). The isolation of such a fascial tube is not difficult except in its postero-medial segment, where separation from the vas calls for some care. The further treatment of the sac, from torsion to final removal, follows accepted procedure. The stump having been allowed to retract, a chromicized gut suture, threaded upon a small Mayo needle, is passed in purse-string fashion around the inner circumference of the fascial tube at its upper end, that is, at the internal ring.

Care must be taken at this stage to avoid picking up the vas or puncturing the veins of the plexus. The tying of this suture so effectively narrows the ring that it once again closely surrounds the structures of the spermatic cord. A few stitches serve to bring the margins of the cremasteric fascia together again, and the operation is completed by suture of the external oblique aponeurosis and closure of the wound.

In recurrent herniæ or those in which the transversalis fascia appears too generally attenuated for repair, I make use of a fascial graft by Gallie's method. It is essential that the graft be not treated as a suture material and drawn tightly; it is simply woven between the lower margin of the internal oblique and conjoined tendon on the one hand and Poupart's ligament on the other. The graft is then spread out by suturing adjacent edges together with fine chromicized gut.

It should not embarrass the internal oblique in its sphincteric function and should play no part but that of a new floor to the canal, embracing laterally the spermatic cord at its entrance thereto. I have been very satisfied with the results of this operation in large, recurrent and direct herniæ alike.

Bacon has written: "In learning where there is much controversy there is many times little enquiry." Reflection upon such cases as those cited has led me to enquire if "the sac, the whole sac, and nothing but the sac" might not be something just less than the whole truth, and as a result of this enquiry to attempt a fuller and more anatomical restoration of the canal than that afforded by removal of the sac alone.

Case Reports.

HYPERPARATHYREOIDISM.

By KENNETH MACKENZIE,
Auckland.

A FEMALE, aged forty-nine years, was admitted to the Auckland Hospital in April, 1934. At the end of 1930 she began to suffer from nausea, which continued for several months. For one month she vomited every day; she was found to have anæmia of the secondary type and achlorhydria. During the illness she noticed tingling pains in the hands and feet, stiffness in the legs and a sense of the leg bones threatening to give way when she stood.

These symptoms continued for eighteen months, when, after a slight fall, a fusiform swelling was noticed at the upper end of the right tibia. In October, 1932, the bone broke as she got up from bed one morning. X-ray examination revealed a large cyst of the tibia at the site of the fracture and a cystic degeneration of a number of the other bones. Since that date she had been bed-ridden and with rest the dyspepsia disappeared and the general health improved. For some months she had painful muscle cramps, but these had almost entirely ceased. In the last few months much swelling of the legs developed, the left leg became deformed and the spine bent forward.

Examination showed that the fracture of the tibia had not united, that the left lower limb lay in the position of a fractured femur, and that there was a considerable degree of dorso-lumbar kyphosis. Great œdema of the legs and albuminuria of moderate amount were present. The thyreoid gland was not enlarged, but a firm tumour could just be felt at its left inferior pole. Radiographs revealed an extreme degree of decalcification and cyst formation in almost all the bones, but most intense in the pelvis, femora and tibiæ. The deformity of the left leg was caused not by a fracture, but rather by a torsion of the fibrous remnant of the femur. There was no sign of calculus formation or of calcification of the kidneys. Renal function tests gave satisfactory results. Figures I and II were taken in April, 1934.

A chemical examination of the blood serum showed the calcium content to be 17 milligrammmes per 100 cubic centimetres, as against a normal of 9 to 11 milligrammes, and the phosphorus content was 2·1, as against 2·5 to 3·5 milligrammes for the normal content. This hypercalcæmia established the diagnosis of hyperparathyreoidism, arising from a parathyreoid tumour—the firm nodule palpable in the neck.

Operation confirmed the diagnosis. To the lower part of the left lobe of the thyreoid gland a firmer mass was closely applied, but was easily dissected free and removed. The measurements of the tumour were 3·75 by 1·8 centimetres (one and a half by three-quarters of an inch), and it was reported to be an adenoma.

On the day following the operation the serum calcium had fallen to 11·7 milligrammes, six days later to 10·5 milligrammes per 100 cubic centimetres. At this time paræsthesia of the fingers was complained of. Calcium was freely administered, but in the subsequent week the serum calcium fell as low as 7·0 milligrammes, and the classical signs of tetany developed.

Intramuscular injections of calcium gluconate were given every day, and after another week the symptoms passed and the level of serum calcium slowly rose

FIGURE I.

FIGURE II.

FIGURE III.

FIGURE IV.

till at six weeks it reached 8·6 milligrammes per 100 cubic centimetres. The general health had then greatly improved, the albuminuria had disappeared, and the œdema was much less. The patient returned to her home in the country. She was able to get out of bed five months later and stand a little after fifteen months.

In September, 1935, she was reexamined. Her general health was very good. There was still œdema of the legs. A considerable amount of fat had been put on. The serum calcium level was at 8·9 milligrammes per 100 cubic centimetres. Radiographs showed disappointingly little recalcification of the bones, especially of the tibiæ.

In May, 1936, she was again admitted to hospital. She was able to walk slowly with assistance, but was much hampered by the deformity of the left femur. There was now a great advance in calcification of all the bones, though cystic spaces persisted. The fracture of the right tibia had firmly united. All œdema had disappeared. The calcium level was 9·8 milligrammes per 100 cubic centimetres of serum. Figures III and IV were taken in May, 1936.

On June 3, 1936, a subtrochanteric osteotomy was performed. The bone was fairly solid to the saw, and its extreme vascularity was a striking feature.

Addendum (November 24, 1937).

It is now eighteen months since the osteotomy was done. Fibrous union only has taken place. There is considerable thickening round the bone. Angulation movement of a few degrees is present at the line of section. Radiograms show some bone formation mesially from the upper part of the bone, but none crossing the fracture.

The general health is excellent. Progress of lime deposit in the skeleton is slow. The patient is getting about in comfort in a walking caliper.

RENAL ABNORMALITY SIMULATING AN ABDOMINAL TUMOUR.

By E. R. Reay,
Christchurch.

The following cases are reported to show the not infrequent occurrence of ectopia and abnormality of the kidney, and the confusion that may arise from an abnormal kidney being in an unusual position. Three facts only in the development of the kidney will be mentioned:

1. The permanent kidney arises from the metanephros, the third and most caudal development of the Wolffian ridge. The primitive position is in the true pelvis opposite the second sacral segment.

2. The ureteric bud from the Wolffian duct approaches the metanephros from the lateral aspect. When union is effected, the renal pelvis is anterior and the calyces face medially. As the kidney ascends to its permanent position in the lumbar region, the organ rotates on a vertical axis through almost 180°. The pelvis becomes posterior, the ureter opens on its medial aspect, and the calyces now look outwards. If renal migration is not attempted (Case I), or is imperfect (Case II), or if fusion anomalies occur between the two kidneys (Cases III and IV), the primitive layout is retained in varying degree.

3. The blood supply of the early kidney is obtained by many vessels from the nearby larger arteries, the aorta, common iliac and others. Not until migration is complete are the vessels collected into a single pedicle, and even then it is not uncommon to find an abnormal vessel at the upper or lower pole. Ectopic or fusion types retain the scattered blood supply arising from neighbouring large vessels.

Four cases of developmental error will be described.

Case I.

A married woman, aged twenty-seven years, who was in her first pregnancy, was admitted to hospital with a diagnosis of obstructed labour. She had had pains for three days, but had made little progress. Vaginal examination disclosed a mass in the pelvis behind the uterus. A Cæsarean section was performed, and a live child was delivered. The pelvic mass was exposed and found to be retroperitoneal. The surface was lobulated, and was covered with large vessels coursing in all directions. It was mistaken for a malignant tumour, and the surgeon made an attempt to remove it. On the anterior surface a structure resembling pelvis and ureter was disclosed, and the attempt at removal was abandoned.

The convalescence was uneventful and the patient was sent for urological investigation. The fixed mass was plainly felt *per vaginam*. Cystoscopy revealed an inward bulging of the bladder wall on the right postero-lateral surface; the left ureteric orifice was absent, with the corresponding half of the trigone. The interureteric bar and trigonal muscle did not extend beyond the mid-line. The right orifice was normal in appearance and showed excellent function with a dye test. The ureteric catheter could be passed for only a few centimetres. The pyelogram revealed all the features of earliest development—the primitive position, the lateral ureter and inward pointing calyces. The opaque fluid in the bladder revealed a filling defect due to the renal mass indenting the full bladder (see Figure I). The diagnosis was single ectopic kidney.

Case II.

A boy, aged twelve years, was admitted to hospital complaining of a painful lump in the abdomen. This was first noted after an injury at football. The lad lived in the country and had previously been healthy. Examination revealed no abnormality except a swelling the size of a large orange, close to the mid-line, in the right side of the lower part of the abdomen. The mass felt soft and cystic, had a limited mobility, and was tender on pressure. Investigation of blood and urine revealed no abnormality. The Casoni test gave a positive reaction,

FIGURE I.

but the result of the complement fixation test was negative. The diagnosis of hydatid was made and the abdomen was opened. The surgeon found a solid retroperitoneal tumour lying over the right sacro-iliac joint. A renal origin was suspected, and the abdomen was closed. The child was later discharged without further investigation.

Some six months later the boy was again sent down. He had been injured again, and this time had a transient hæmaturia. The local condition in the abdomen was as before. Cystoscopy revealed a normal bladder appearance. The result of the dye test was a little poorer on the right side. The right catheter passed only a short distance. Pyelograms revealed a normal left kidney, but the right kidney was found to be situated opposite the sacro-iliac joint (Figure II). Rotation was half complete, the calyces at this stage looking in an antero-posterior direction. This position, with the convex renal border facing anteriorly, made

the kidney more prominent in the abdomen. Further operation was decided upon to attempt to place the organ in a less obtrusive position.

The abdomen was opened by an incision through the lower right rectus. The cæcum was reflected inwards and the kidney was exposed. The convex border faced the anterior abdominal wall, and along the concave margin from pole to pole were a series of vessels arising from the aorta and common iliac arteries. Because of this vascular anchor, it was impossible to lift the kidney. The vessels were carefully dissected free and the shorter ureter was mobilized. The rotation of the kidney could then be completed. The posterior surface was decapsulated

FIGURE II.

and the organ was fixed by suture to the iliacus and quadratus muscles. Care was taken to avoid pressure on the ureter by the vessels at the lower pole. A vein crossing the ureter was cut between ligatures. The caecum was replaced and the wound was closed with a stab drain in the loin.

The result was satisfactory in so far as there was now no prominent abdominal tumour, although the kidney was readily palpable on the back wall of the iliac fossa. Seen two years later, the boy had had no further trouble, and the kidney was firmly fixed. These two cases show the difficulty in attempting to move an ectopic kidney to a more favourable location. The short ureter and wide vascular attachments render mobilization almost impossible. One hesitates to remove such a kidney when function is good.

Case III.

A single man, aged forty-five years, complained of a painful lump in the right side of the upper part of the abdomen. There had been a constant dull

pain in the right side and recurrent attacks of acute colicky pain radiating to the groin. There was no history of jaundice or of urinary disturbance. The patient had had three abdominal operations without relief. The symptoms had been in evidence about fifteen years.

FIGURE III.

The patient was a sallow, ill-nourished adult. The abdomen showed the scars of his previous operations. Filling the right upper quadrant was a hard mass, apparently continuous with the liver above. There was no area of resonance over it, and it was not mobile. Clinically it felt like an intraperitoneal tumour, possibly connected with the liver. In view of the previous unsuccessful operations, a complete study of the urinary tract was advised. The cystoscopic view of the bladder showed each ureter to be in normal position. Function on the right side was normal. On the left side urine appeared in a regular drip without

peristaltic effort, and the appearance of the dye was much delayed. Each ureteric catheter passed without difficulty, but some five centimetres short of the usual distance. The left catheter released a free flow of clear urine under increased pressure. A plain radiogram showed the right catheter swinging outward, and the left crossing the mid-line to reach the right side. A double pyelogram revealed the unusual fusion anomaly, unilateral fused kidney. There was a filling defect of the left pelvis. It will be seen (Figure III) that the pelves retained their anterior position and that the calyces of each segment faced each other. The lower portion of the left pelvis was filled with iodide and appeared normal. The upper portion showed the iodide diffused in poor concentration, suggesting a hydronephrotic sac. The likely cause of a partial hydronephrosis, such as this, is an aberrant vessel crossing the pelvis. This was the diagnosis before operation.

The abdomen was opened by an incision through the right rectus muscle. The ascending colon was reflected with difficulty from a large mass occupying the upper quadrant. The surface of the tumour was covered with very large vessels. Lobulation was present, but no division could be traced between the two kidneys. Toward the mid-line on the anterior surface a distended sac was found. When traced down, this was found to terminate in the left ureter. The sac was tightly bound down below by a large vein crossing from the right renal segment to the *vena cava*. When this vessel was cut the pelvis readily emptied. The region of the ureto-pelvic junction being normal, no plastic repair was attempted. No attempt was made to isolate the right pelvis or ureter. The colon was replaced and drainage was provided through a stab wound in the flank. Convalescence was without incident. This patient's condition was very much improved, although there remained some discomfort from the large fused kidney.

Case IV.

The chief complaint in this case was gradually increasing frequency of micturition, extending over a period of fourteen months. The patient, who was a married man, aged thirty-five years, stated that during the previous six months he had been treated without success with various urinary antiseptics. He had lost weight and strength. The frequency had become much worse, and was associated with tenesmus. The patient looked ill, and was sallow and wasted. On examination there was found in the left hypochondrium a hard fixed mass with an irregular surface. It could not be felt in the loin. There was no band of colon resonance crossing the tumour. The urine was strongly acid, contained profuse pus, and was sterile on culture. No tubercle bacilli were found in ordinary specimens.

Cystoscopy was difficult owing to the extreme spasm and contraction of the bladder. The vesical mucosa was generally inflamed with superficial ulceration. The left ureteric orifice was retracted and gaping. It was surrounded by breaking down tubercles, and the efflux was cloudy. The right efflux was clear, and dye appeared in good concentration in five minutes, while on the left side it was faint in ten minutes. A double pyelogram revealed a typical tuberculous appearance in the left segment of a horse-shoe kidney (Figure IV). The pelves were low in position, close to the mid-line, and the calyces faced medially. No indication of the commissure could be seen in the radiogram. The right ureteric specimen of urine was normal, and the left contained pus and tubercle bacilli.

At operation a lumbar incision was employed, not extending very high posteriorly. Anteriorly it was brought well over the iliac spine to expose the commissure. The upper pole of the left segment was freed from dense adhesions. Vessels were seen to enter the concave border from pole to pole. The vascular connections were severed from above downwards as they were exposed. The thickened pelvis lying anteriorly was demonstrated. The ureter crossed in front of a thick bank of commissural tissue. It was traced well down in the bony pelvis and divided. This freed the left segment except for the commissure, which was solid renal tissue showing no line of demarcation. The pyelogram showed no calyx from the left entering the commissure, although one from the right

extended well into it. The area was well packed off with sponges. Clamps were applied to the mid-point of the connecting band, and it was divided with the diathermy needle. A little purulent urine escaped into the packs. The section had entered a tuberculous calyx. A further wedge was taken reaching normal tissue, and the stump was closed with mattress sutures. Drains were placed to the stump and in the renal fossa, and the wound was closed. The

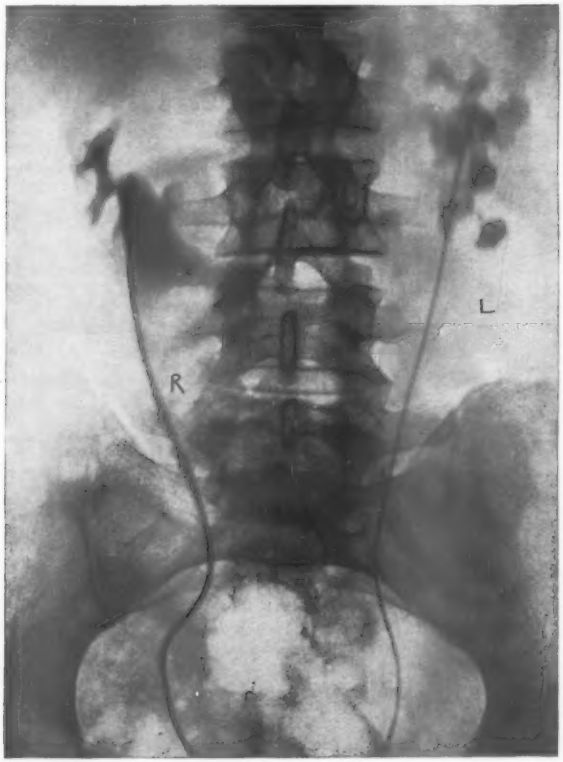


FIGURE IV.

wound healed cleanly, but some three weeks after operation a sinus formed at the site of drainage. This discharged thin pus for three months.

Six months after operation the wound was sound and the patient had gained three stone in weight. The urine contained only a few pus cells. The bladder capacity was increasing. One point of interest was the presence in the commissure of a diseased calyx, although there was no indication of it in the pyelogram. The inferior calyx on the sound side was clearly seen entering. The explanation was the filling of the infected bottom calyx with purulent *débris* preventing the entrance of the opaque solution.

The two last cases show how the fusion type of anomaly may cause confusion in abdominal diagnosis. The fused kidney seldom completes its ascent does not reach the renal fossa, and especially when the seat of pathological tumefaction occupies a prominent position in the abdomen.

Summary.

This small series of cases illustrates:

1. The not uncommon occurrence of renal abnormality.

2. The relative frequency with which the abnormal kidney is the seat of a pathological condition.

3. That an abnormal kidney may be mistaken for an abdominal tumour.

4. The value of urological investigation in the presence of any unusual type of tumour discovered on abdominal palpation.

The Australian and New Zealand Journal of Surgery.

All articles submitted for publication in this journal must be typewritten and double or treble spacing should be used. Each article should conclude with a brief summary and statement of conclusions. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without any abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal together with that of the journal in which the abstract has appeared, should be given, with full date in each instance.

When illustrations are required, good photographic prints on glossy gaslight paper should be submitted. Line drawings, charts, graphs and so forth should be drawn on thick white paper in India ink. Authors who are not accustomed to prepare drawings of this kind, are invited to seek the advice of the Editor if they are in any doubt as to the correct procedure. Skiagrams can be reproduced satisfactorily only if good prints or negatives are available.

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A GORDON CRAIG LIBRARY SERVICE.

THIS year the Council will receive the endowment which the late Gordon Craig provided for the Royal Australasian College of Surgeons. A foundation Fellow of the College, Gordon Craig was very anxious that one of its main functions should be the teaching of surgery in its highest form; and he held that this teaching should concern itself not only with the education of medical men who wish to become competent surgeons, but also with the continued education of Fellows, so that they might be able to keep abreast of all advances in surgery and make the best surgical skill available for the people. The Council feels that there can be no better method of educating its Fellows than by making available to them, by a library service in their widely separated homes throughout Australia and New Zealand, all the resources of modern surgical literature. As a permanent memorial to one of its Fellows who has by a statesman-like action helped the Royal Australasian College of Surgeons to carry out these functions for which it was designed, the Council will name the library attached to the College building in Melbourne The

Gordon Craig Memorial Library; and in connexion with this the library service will then be established.

The present library is fully stocked with current literature, and contains a considerable number of recent surgical works; these, with other books, reprints and periodicals which will be rapidly added to it, will form the basis for The Gordon Craig Memorial Library. Each work in the library will contain the late Gordon Craig's bookplate, with which will be combined the Achievement of Arms of the College. The Council will design this library service so that it will come within the reach of every Fellow of the College. In this way it hopes eventually to build up the best reference library of surgical literature in the southern hemisphere.

The library will be under the direction of Mr. Robert Syme, and will be administered by the Executive and the Secretary of the College, Mr. Wheeler. When the necessity arises the Council will appoint a librarian, who will have a good knowledge of French and German, as well as of English, and who will be capable of making photostat copies or of gathering the literature on a particular subject.

The surgical literature available for Fellows will comprise: recent books, mostly monographs on subjects; current periodicals; reprints; collections of literature and references on special subjects, a series of which will be gradually built up as this library service develops. As each research into literature on special subjects is made at the instance of a Fellow, it will be duplicated and filed for the benefit of future investigators of the same subject. The Fellows will be kept in touch with incoming literature in several ways. A monthly library circular will contain a list of books received during the month, and the titles of the more important original articles in the current literature of the month. THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY will contain an amplification of the present foreign literature section, in which specially selected articles taken from foreign surgical journals are fully abstracted. An exhibition of recent books and recent current literature will be made at each annual meeting.

This library service will be available either at the library at the college headquarters, or by loan on personal or postal application for a book, a current periodical or a research into literature. Where a Fellow desires to have for reference a particular journal that he has consulted, the library service will, if possible, procure it for him and supply it to him at cost. A charge will be made for all journals and periodicals not returned by a certain time; and if the literature or its value is not

returned within a certain time, the Fellows will be struck off the list of those entitled to library service. Special arrangements will be made for the return of books and periodicals from different parts of Australia and New Zealand.

A successful library service will live and prosper if there is a demand for it. A demand for it will arise if Fellows will report their experiences and write articles in THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY for the benefit of their brother surgeons. A demand for a library service will also arise if Fellows will use the library for the main purpose for which it is created by the College, that is, to enable them to continue their education by keeping abreast of advances in surgery, so that they will be able to give to their patients the best surgical service that is possible.

For the Executive,

H. B. DEVINE,

Chairman.

Surgery in Other Countries.

[In this column will be published short résumés of articles likely to be of practical value from Journals published in other countries and not readily accessible to surgeons in Australia and New Zealand.]

A New Method of Treatment of Subacute and Chronic Abscesses of the Cerebral Hemispheres.

Clovis Vincent, Marcel David and Harden Askenasy: "*Sur une méthode de traitement des abcès subaigus et chroniques des hémisphères cérébraux*", *Journal de chirurgie*, Volume xlix, Number 1, January, 1937.

In a most interesting communication, including the detailed histories of five cases, the writers present a method of surgical attack on brain abscess not hitherto generally employed. The essential features are as follows: Operation is delayed for a period of weeks or months in order to allow pus to be encysted in a thin envelope. An osteoplastic flap is raised as for a tumour; the *dura mater* is not incised, but decompression is produced by simple elevation of a bone flap of large size. The pus is aspirated by means of Cushing's brain needle, and the aperture in the *dura mater* is sealed by diathermy. (Aspiration may be repeated several times.) The scalp wound is completely closed without drainage, the cut bone flap being left *in situ*.

Later the skull is reopened. The dura is incised, and careful dissection of the abscess cavity from the surrounding brain is carried out by the diathermy knife, the wall of the abscess being removed as a solid tumour. Sealing off of the site of entry of the infection is carried out by application of a strip of *fascia lata*. (This site is the frontal bone in the case of frontal sinusitis, and the mastoid area when the latter is the primary focus.) Complete closure of skull by bone flap and scalp without drainage is carried out.

It is to be noted that acute cerebral abscess is characterized by an enormous œdema; frequently a whole cerebral lobe is involved. The pus is not the cause of the symptoms, but rather the brain œdema and the toxic effects of the infection quickly kill the patient. After the third week a capsule develops and gradually thickens in a patient who survives. The œdema subsides and the encysted pus can be regarded as producing symptoms in a manner similar to a brain tumour. Vincent and his colleagues state that aspiration and drainage are effective only when the abscess is small and presents close to the surface of the brain. Most surgeons will agree with the authors that the results of drainage, even with the modern methods, are disappointing in many cases. The soft brain soon blocks the drain, and infection of the meninges is an all too frequent complication. Few can show a series of recoveries comparable with those of Macewan or Coleman. The methods of the last named are probably applicable to the smaller abscesses associated with mastoid infection. The larger chronic abscesses, more deeply situated, present a problem which the method of Vincent appears to solve.

The following *résumé* of the case histories of the five cases reported will indicate briefly the method employed:

A girl, aged fourteen years, complained of periorbital œdema in October, 1934. Six days later she had headache, intense above the left eye, and vomiting, but no fever; her general condition was good. Paroxysms of severe headache were followed by somnolence and torpor, asthenia and loss of weight. About November 10, stiffness of neck, bradycardia, ptosis of the left upper lid, dilatation

of the pupil and blindness of the left eye occurred. Left facial paralysis was noted, and the patient complained of headache and neck pains. Bilateral papillœdema, more marked on the left side, was present. Lumbar puncture yielded clear fluid under increased pressure; the fluid gave no cellular reaction, the albumin content was 0·40 and the glucose content was 0·50; the fluid contained no organisms. Somnolence increased, cerebral cry, pallor, mental changes, incoherence were noted. There was no incontinence of urine or fæces. Head retraction occurred. The patient was then admitted to the neurosurgical wards. She could not cooperate. Her cutaneous reflexes and tendon jerks were unaltered. Right facial paresis and tenderness in the left temporal region were present. Bilateral ventricular puncture was performed, air being injected into the right ventricle. X ray examination showed no air in the left ventricle, the right ventricle was pushed over to the right, the third and fourth ventricles were deviated to the right. A diagnosis of left frontal tumour was made.

Operation was performed on November 23 under local anæsthesia. A left frontal flap was elevated; the *dura mater* appeared stretched and pale. The frontal horn was sought, but not found. The dura was opened by a tiny incision; a puncture was made by a trocar and at a depth of four centimetres a resistance as of a cyst was noted. Twenty cubic centimetres of pus were withdrawn. It was decided not to open the abscess and not to drain it. A piece of muscle was placed over the dural opening. The bone flap was replaced, and the scalp was sutured in two layers. Improvement in the patient's general condition followed, vomiting and headache disappeared, and appetite returned. Despite this improvement, the girl was agitated and tended to handle the wound. Further puncture was performed on December 10. The papillœdema disappeared. A second operation was performed on January 7, 1935. The flap was elevated through the old incision. The dura was thin and non-adherent. A longitudinal incision of the dura was made; the frontal lobe was incised at a depth of two centimetres; the wall of the abscess was found. Dissection of the abscess wall was carried out by diathermy and sucker. When it was almost completely freed the index finger aided the removal of the abscess cavity in one piece without its being opened. The vessels were coagulated. Bits of muscle were placed where required for hæmostatic toilet, and a piece of *fascia lata* was applied over the region of the frontal sinus. The bone flap was replaced after complete suture of the dura. The scalp was sutured in two layers without drainage. The abscess weighed 150 grammes. It occupied the whole of the orbital part of the anterior fossa as far back as the lesser wing of the sphenoid.

After operation, cerebral irritation was present for four days, but no more than is usual after a frontal tumour operation. Local reaction was pronounced; the flap was elevated by fluid and blood, which was aspirated daily; œdema of the eyelid and forehead was present. By January 24 the eye grounds were normal; there were no objective nervous signs. The patient's general appearance was normal. In October, 1935, the girl was in good health, and was engaged in her usual employment.

A case of temporal lobe abscess was dealt with as follows: A child, aged thirteen years, had a running left ear on February 1, 1935. The course was the usual one for an acute middle ear inflammation. On February 10 a meningeal syndrome developed—high fever, Kernig's sign, neck rigidity, vomiting and photophobia. Lumbar puncture revealed an intense leucocytosis, but no organisms. An aurist decided that mastoid operation was not indicated, as there was no local reaction in the mastoid. Medical treatment was advised, that is, "Pyoformine", "Collargol Septicemine" into the spinal canal and fixation of the abscess. After a stormy course, during which the child developed spasm of the right side of the lower part of the face, left temporal and left orbital pains, the condition seemed to improve until June 15, when generalized convulsions and confusion were noted. Loss of weight and intense left temporal headache occurred, with crises of melancholy, during which the child kept repeating: "I know I am going to die, but I do not want to die." There was marked papillœdema; the visual field was normal. No leucocytosis was present. On August 15, 1935, neurological examination disclosed an anxious, hostile child, crying on being handled. The child's posture

was normal, no loss of equilibrium was present, slight right facial paralysis was noted, with slight aphasia (difficulty in finding certain words in conversation). The X ray findings of the mastoid region were normal. Ventriculography was performed. The left ventricle was not found; the right ventricle was punctured and 25 cubic centimetres of clear fluid were withdrawn; 15 cubic centimetres

FIGURE I. The abscess has been removed as a single mass without opening of its wall. The weight of the abscess was 150 grammes.

FIGURE II. Case III (not reported in the summary herewith). Decompressive flap without opening of the *dura mater*. Note the considerable elevation of the bone flap.

FIGURE III. Note the adhesions of the anterior surface of the abscess wall to the thick *dura mater* in the floor of the temporal fossa.

FIGURE IV. The abscess removed as one piece. Its weight was 120 grammes.

of air were injected. The ventricular fluid showed a slight reaction—five to six lymphocytes per cubic millimetre and 0·45 gramme of albumin *per centum*. A ventriculogram showed deviation of the ventricles to the right; the left ventricle was flattened.

Operation was performed on August 16, local anæsthesia being used. A large elliptical flap with the base anterior was lifted so as to expose the temporal lobe completely. Puncture of the temporal lobe with a Cushing trocar disclosed an encapsuled abscess, the wall of which was sufficiently resistant to permit of its complete extirpation. The temporal lobe convolutions were flattened and greyish-yellow. A transverse incision of the middle of the second temporal convolution

was made, and at a depth of one centimetre the wall of the abscess was found. By sucking away the soft brain tissue surrounding the abscess the incision was enlarged. The abscess wall was as thin as cigarette paper in its posterior part; in front it was much thicker and bilobed. Adhesions to the lesser wing of the sphenoid and to the floor of the temporal fossa were separated with difficulty, an enlarged posterior temporal vein causing trouble. The *dura mater* was almost one centimetre thick in places and very vascular. Bleeding points were coagulated. The *dura mater* was completely closed, the bone flap was replaced, and the wound was closed without drainage. The abscess weighed 120 grammes. Examination of the pus revealed no organisms. The post-operative course was excellent. No adhesions, no fever, no œdema of the face and no leakage of cerebro-spinal fluid occurred. The child was up on the fifteenth day, quite normal in every respect.

The brilliant results obtained by the authors should inspire neurosurgeons to extend their field of operations, and should also encourage otologists in dealing with these hitherto extremely difficult cases. The long delay between the initial onset of the disease and operation certainly prepared the stage for the neurosurgeon. It is doubtful whether more than a few patients, suffering from a relatively benign infection, would tolerate such delay and remain suitable subjects for neurosurgical attack. It would appear that the "heroic" conservatism followed by the radical methods of the Vincent school bid fair to bring purulent collections in the cranial cavity into line with similar conditions in the pleural and pelvic cavities.

Further communications on the subject will be welcomed. Meanwhile the authors are to be congratulated on their remarkable achievements in an admittedly hazardous field of surgery.

A. E. COATES.

ARTHRODESIS OF THE KNEE JOINT FOR TUBERCULOSIS IN CHILDREN.

Jean Calvet: "*Valeur de l'arthrodèse du genou dans le traitement de la tumeur blanche du genou de l'enfant et de l'adolescent*", *Journal de Chirurgie*, Volume xlviii, November, 1936, page 646.

THE author reviews at length in a thesis the indications and numerous operations for arthrodesis of the knee joint in the treatment of tuberculous arthritis of children, and appends a full international bibliography. The first point to be decided is the maximum benefit which the classical orthopædic (conservative) treatment can offer at any particular stage of the disease. Certain authors place fervent trust in climatic and institutional treatment directed towards obtaining a healed but mobile joint, but a greater number are those, such as Hibbs, Putti, Sorrel and others, who see such treatment in a bad light. These hold that certain cure with conservation of mobility is the exception rather than the rule, except in the youngest children. The author has been often struck, at the maritime hospital of Berck, by the large number of children returning with complications or recurrences of the disease after some months of normal life. The form of tuberculous arthritis known as *la tumeur blanche* is a serious one, for which conservative treatment can hold out no certainty of cure. Nevertheless, it does not constitute an absolute indication for arthrodesis because in a sufficient number of cases the disease is arrested by conservative treatment; if this fails after three years, however, then operation is indicated. Such a policy has the advantage that the operation is not performed at a dangerous stage of the disease when bacillæmia and much local activity are present. Furthermore, by waiting such a long time an opportunity for spontaneous cure or even for a mobile joint is provided. The early proliferative or fungous stage is a contraindication to intra-articular arthrodesis, whilst the presence of sinuses or abscesses prevents extra-articular arthrodesis. The exact indications for operation in children occur in the chronic and recurring forms, or when much deformity and displacement are present. The large number of methods for arthrodesis of the knee joint is surprising. The author is very unfavourable to resection of the knee joint in children, and favours either subcutaneous intraarticular pegging or the use of an extraarticular tibial bone transplant. He sets out his reasons as described below.

Resection of the Knee Joint.—In patients above the age of fourteen years resection gives excellent results, but difficulties arise. They are pseudarthrosis, gradual softening and bending of the tibio-femoral fused block of bone, or deformity due to interference with the epiphyseal plate of growing cartilage. Therefore, most surgeons supplement fusion with some form of internal graft. Rowley Bristow and Girdlestone use a central peg; Waldenstroem uses his *os novum*, Trèves uses powdered bone, and so on. All these palliatives complicate the operation, and suggest that it is preferable not to resect the knee joint in the young child, unless pronounced deformity is present. Whilst Calvert admires the large number of tuberculous knee joints benefited by the operative method of resection sponsored by Putti (Figure I) and described by Girardi, nevertheless he criticizes the principle of the operation because a large arthrotomy and division of the soft parts may be followed by constitutional and local reactions. Furthermore, in a child the segment of the tibia used as a graft includes a portion of the upper epiphyseal plate of cartilage. According, therefore, to the author, only two proceedings can be recommended before the age of resection. One is intraarticular and the other is strictly outside the joint capsule as described below.

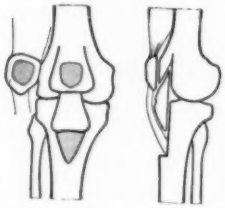

FIGURE I.

Intraarticular Arthrodesis (Richard).—The intra-articular arthrodesis of Richard consists of trans-articular pegging which respects the epiphyseal plate of cartilage (Figure II). There is no arthrotomy because the bone peg (cut from the tibia, and this is really the major part of the operation) is driven from the femoral condyle through the joint line obliquely into the tibia. A small incision is made over one or other condyle and then the bone is drilled. An important pre-requisite for the bone pegging is a preliminary X ray examination of the knee joint to which is strapped a metal band (Figure II, A) marked off in centimetres.

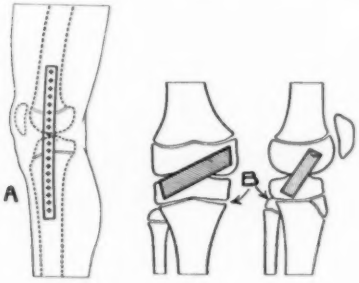

FIGURE II.

FIGURE III.

Pictures are made in two planes with the ruler strapped accordingly. After some calculation, this enables the distance of the epiphyseal lines from the joint line to be measured. The length of bone peg required is also determined. A plaster is applied for three months, and then a posterior gutter is cut to permit heliotherapy. By this slight operation the transarticular graft assists in the reparation of the disease by fixing solidly the joint, it supplies calcium and, by perforating the articular cartilage, creates an irritation in favour of ankylosis. Apparently the bone peg can survive the tuberculous disease and function as an osteogenetic nucleus for the production of bony ankylosis. This appears plainly in Figure III, which shows the condition of affairs before and after the trans-articular pegging. Note the splendid bony ankylosis obtained by this simple

method. In seventeen cases reported by the author there were only two failures, and they were due to a mistake because the bone peg had failed to cross the joint line from the femur into the tibia, and therefore had not engaged the opposing articular surfaces. Also Girardi reported one case of fracture of the graft, but it did not prejudice the ankylosis.

Extraarticular Arthrodesis (Figures IV and V).—De Delahaye employs a long tibial bone transplant, traversing a groove in the patella and attached at one end in the femur through the quadriceps muscle and at the other end in the tibia. The method is recommended for children of between eight and fourteen years of age, when the disease has been long drawn out. It is also recommended in the recurring or relapsing cases, especially as an intraarticular procedure is feared in the presence of an active focus. Massart uses a maternal graft. The use of pegs of *os purum* is still being studied. Pseudarthrosis of the transplant is serious, and is due to insufficient contact at either end. Fracture of the tibial span is rare because there is little strain on it. The exact function and effective purpose of it seem a little uncertain, but since it is used in infants the slender bony ankylosis obtained so far from the joint appears to be satisfactory, and it usually grows *pari passu* with the bones of the limbs.

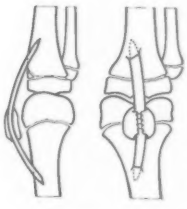

FIGURE IV.

FIGURE V.

In conclusion, the author points out that scrupulous care is required in selecting cases suitable for arthrodesis in children as compared with adults; care is also needed in the technique selected. Otherwise the legitimate efforts of those directed towards ameliorating the prognosis of tuberculosis of the knee joint in children are likely to be submerged under the number of failures due to unwise operations ending fatally.

THOMAS KING.

REDUCTION OF OLD SHOULDER DISLOCATIONS UNDER LOCAL ANÆSTHESIA.

H. Tietze, Leipzig: *"Unblutige Reposition veralterer Schulterluxationen mit Hilfe von Novocaininjektionen", Der Chirurg*, August 15, 1936.

TIETZE commences his article with the statement that experience shows that the end results of open reduction of old-standing shoulder dislocations leave much to be desired, and that intensive mechanico-medical after-treatment has not done much to improve them. Therefore, he reports a method of treatment originally devised by Payr, by which it is possible to reduce almost impossible-looking dislocations; and which has the merit of simplicity and of sparing the already mishandled joint from further insult. It has, however, occasionally failed, and should not be used when there is interposition of soft tissues, or when there is a fracture of the humeral head.

The theory of it is that the hindrance to reposition is due, not to any mechanical bar in the way of ligaments or joint injury, but purely to the development of a condition of hypertonic contracture of the muscles surrounding it, and particularly of the subscapularis. This is based on work done by Payr, Dollinger and others.

The procedure laid down as routine at Payr's clinic at Leipzig is as follows: The capsule is filled, and the general shoulder musculature is infiltrated, with 0·5% "Novocain", especial care being taken to infiltrate the subscapularis thoroughly. Adrenaline is not used with the "Novocain". Then reduction is attempted under anæsthesia. If this fails, no more forcible measures are used, and the patient is treated by "Novocain" injections every two days, with careful stretching of the muscles. The hard tension is thus relaxed, the head can be rotated, and the arm abducted to 60°. Eight days later a fresh attempt at reduction is made, and this is generally successful. In the after-treatment prolonged immobilization is deliberately avoided. After two days, "Novocain" is again injected, and active movement exercises are begun along with massage and the interrupted current. With this treatment the joint becomes free from pain and the muscles recover, ensuring a very satisfactory end result.

It is stressed, however, that an immediate good result must not necessarily be expected. The first aim is to loosen the muscles and the capsule, and to combat the contracture. Prolonged preliminary treatment is often necessary. "Novocain" injections must often be repeated several times before manœuvres for reposition are successful, sometimes after several fruitless attempts. It is necessary, therefore, to carry the treatment out according to rule, and to persist over the necessary length of time, before deciding to change over to operative methods.

ARTHUR E. BROWN.

SERUM THERAPY FOR APPENDICITIS AND PERITONITIS.

Dr. W. Nobel, Basel: *"Über die Serumtherapie der Appendicitis und Appendicoperitonitis"*, *Der Chirurg*, October, 1936.

IN gangrenous and perforative appendicitis and its ensuing peritonitis, the main bacteria to be reckoned with are gas gangrene bacilli, which are second in number only to *Bacillus coli*; and with them are found streptococci and enterococci. The great danger of the gas gangrene bacillus lies not in its gas-forming properties, but in its toxicity. General intoxication occurs early, leading to paralysis of the vasomotor centre and circulatory collapse. Serum therapy, therefore, becomes in principle a question of combating this general toxæmia from the anaerobes. The special advantage of serum therapy lies in its detoxicating property; and the effectiveness of this depends on four factors: the nature of the antitoxin, the time of its administration, the dose given, and the method of administration.

Taking the last point first, intravenous injection is the most effective, intraperitoneal injection next. The essential thing is to get a high concentration of antitoxin into the blood as soon as possible. Working with diphtheria antitoxin, Berghaus has shown that the intravenous route is 500 times as potent as the subcutaneous route, and 90 times as effective as the intraperitoneal. On account of several reported fatalities from anaphylaxis due to using the intravenous method, this route has been largely given up; but quite wrongly, because by suitable dilution and by very slow administration, and also by giving the first dose under an anæsthetic, the dangers may be safely avoided. A large intravenous dose is therefore given in bad cases, while in slighter infections an intramuscular injection is given. Given subcutaneously, its effect is so small that it is not used in that way.

The dose used will, of course, vary with the inclination of the surgeon, the nature of the case, and the condition of the patient. Nobel gives 10 to 40 cubic centimetres intravenously immediately after the operation, along with an intramuscular or intravenous infusion of Ringer's solution or glucose. This procedure is repeated after twelve, twenty-four and forty-eight hours. He has not used the intraperitoneal route extensively. Many failures to derive benefit have been due to too small a dosage.

The serum used is Weinberg's *"Sérum antigangréneux"*, which is antitoxic to the Welch-Fraenkel gas gangrene bacillus, the *Vibrion septique*, *Bacillus histolyticus*, *Bacillus œdematiens*, and *Bacillus sporogenes*, though lately the

Bacillus sporogenes has been omitted. It does not seem essential that all the causative bacteria should be directly attacked. Often in a mixed infection, such as this is, it is sufficient to upset the symbiotic conditions which are necessary for the combined working of the various bacteria. A simple polyvalent gas gangrene antiserum, therefore, has a rational place in the treatment; but the use of a mixed vaccine such as Weinberg's is more reasonable.

The importance of the time factor in administration is very great. The toxic process which shows itself in the reaction between body cells and toxin, has two stages, the second one passing into cell-death. During the first stage the combination of cell with toxin is still reversible, but the cell's function and vitality are damaged, though not irreparably, by interference with its metabolism. During this phase large doses of antitoxin will save the lives of some patients. Later, when the second stage has become very widespread, and a large proportion of body cells have passed into cell-death, neither serum nor any symptomatic treatment will be of any use. This theory is based on experimental work with tetanus and diphtheria antitoxin on animals, and it explains the facts observed in appendiceal peritonitis. The surgeon should not, however, adopt too fatalistic an attitude and withhold serum even from those who are suffering from late infections.

The statistical results are difficult to compare with those of other workers owing to the very great variation in clinical standards. But, taking the severe cases of diffuse peritonitis in Nobel's own clinic, before and after the general adoption of the routine use of serum, the mortality has fallen from 57% to 37%. His detailed statistics are based on too small numbers of cases to be important, but he is firmly of the opinion that something definite has been achieved by its use. When it fails, the four questions to be investigated are: "Was the dose large enough?" "Was the serum specific?" "Was the treatment given early enough?" "Did the patient have sufficient resistance?" The first three factors have been discussed in his paper. The last must remain largely an unknown quantity.

ARTHUR E. BROWN.

Reviews.

THE HISTORY OF GYNÆCOLOGY AND OBSTETRICS.

Clio Medica: A Series of Primers on the History of Medicine. Edited by E. B. KRUMBHAAR, M.D. XVII: Gynecology and Obstetrics, by E. M. JAMESON, M.D.; 1936. New York: Paul B. Hoeber. Foolscap 8vo, pp. 185, with illustrations. Price $1.50 net.

GYNÆCOLOGISTS and obstetricians have long waited for a concise history of their specialty. This want is at last supplied in good measure by Dr. Jameson's "Gynecology and Obstetrics", which forms one of the latest additions to the series "Clio Medica" published by Mr. P. B. Hoeber, of New York.

Up to the present very little has been written in English on the history of obstetrics or gynæcology. The searcher for historical facts has had to hunt through back numbers of medical journals. An Australian author, in the person of Dr. Stewart McKay, wrote some thirty-six years ago "The History of Ancient Gynæcology" and more recently Dr. H. R. Spencer has given us an account of "British Midwifery from 1650-1800"; but we still await a complete and authoritative history of midwifery and gynæcology in English which will compare with the ponderous tomes of the German historians, Siebold and Fasbender.

In the one hundred and fifty pages at his disposal, Dr. Jameson has given us a very concise and interesting account of the progress of the obstetrical art. He deals first of all with midwifery amongst primitive peoples. It is strange to read that these peoples practised cephalic version long before the time of Hippocrates, and made use of various mechanical contrivances, such as whale-bone

loops and silk nets, to assist in the extraction of the infant's head. The story of how the primitive squatting position in childbirth was replaced by the obstetric chair, and this in its turn by the adoption of the prone position, makes interesting reading.

It is well pointed out that lack of accurate anatomical knowledge of the female pelvis hindered the advance of midwifery and gynæcology during many centuries, until the studies of Vesalius in the early years of the sixteenth century made possible the development of scientific midwifery. This growth in anatomical knowledge naturally led to the improvement of surgery, and ultimately to the birth of that branch of surgery which we nowadays call gynæcology. With the seventeenth century began the modern period. To this century belong the great names of Francois, Mauriceau, Deventer and Portal. They fought the great fight against the ignorant midwives, who, entrenched strongly behind the ramparts of female modesty and established custom, had monopolized the care of the parturient woman. The eighteenth century brings us the Scotchmen, William Smellie and William Hunter. Smellie fought the final and successful engagement with the midwives, and after his time it was no longer immodest for a male obstetrician to attend a woman in childbirth.

A most interesting chapter is devoted to the Chamberlens and their invention of the midwifery forceps. A valiant attempt is made to disentangle the various members of this extraordinary family, who delighted in the names of Hugh and Peter, which names they duplicated, a peculiarity that has caused much confusion to medical historians. The story of Hugh the Elder has quite a modern flavour. Evidently tiring of squabbles with his fellow practitioners and of attending the wives of royal personages, he embarked on a career of company promoting. He founded a Land Bank which he forecast would "make England Rich and Happy". The shares being fully subscribed, Hugh departed hurriedly for Holland, where he spent his remaining days, and, incidentally, disposed of the secret of his invention to a Dutch obstetrician.

The tragic story of puerperal fever and of the martyrdom of Semmelweiss is well told. We hear how Oliver Wendell Holmes did his part in showing that child-bed fever was due to infection carried in many cases by the medical attendant, and we realize how stony is the path of the man who wishes to introduce reforms into medical practice.

The book closes with a chapter on gynæcological surgery, a chapter which shows the great debt this branch of surgery owes to American surgeons. The names of Ephraim McDowell, Marion Sims, Thomas Emmet and Howard Kelly are prominent in the history of surgical procedures for the relief of suffering women, and at every step their innovations were opposed and bitterly criticized.

This little book is well illustrated and attractively produced. The busiest obstetrician and gynæcologist will find after reading it that he has gained a wider perspective and a sounder knowledge of his profession.

PULMONARY HISTOLOGY.

The Lung. By W. S. MILLER; 1937. London: Baillière, Tindall and Cox. Royal 8vo, pp. 223, with illustrations. Price: 34s. net.

PROFESSOR W. S. MILLER has been so universally recognized as an authority on lung morphology that his book on the lung is indeed a welcome publication. As stated in the preface, it is the result of his labours on lung structure over almost half a century. It is really a book on pulmonary histology. The concise and decisive text and beautiful illustrations will be of the utmost value to teaching schools and all interested in the morphology of the lung. His description of the lobule as the lung unit is universally accepted. It is noteworthy that he is absolutely convinced that the alveoli are lined with a continuous epithelium, and the illustrations of this being lifted off the wall by an accumulation of serum seem unchallengeable. The reaction to this of B. M. Fried, the protagonist of the histiocyte lining, would be interesting.

Professor Miller has not found those islands or plaques of lymphoid tissue situated beneath the epithelium in the bronchi which Krause described and stated that they formed starting points for bronchiectatic lesions. The chapter on the innervation of the lung by Professor Larsell, of the University of Oregon, who has done so much important work with Fenton on the spheno-palatine ganglion and the nasal mucosa, is in keeping with the standard of the book.

An historical sketch, with illustrations of the work of the old masters from Malpighi to Kolliker, and an excellent bibliography round off this work, which must be deemed an outstanding publication.

OPERATIVE SURGERY.

Operative Surgery. By J. S. HORSLEY, M.D., LL.D., F.A.C.S., and I. A. BIGGER, M.D., with contributions by C. C. COLEMAN, M.D., F.A.C.S., J. S. HORSLEY, junior, M.D., A. I. DODSON, M.D., F.A.C.S., and D. M. FAULKNER, M.D. Volumes I and II, Fourth Edition; 1937. St. Louis: The C. V. Mosby Company. Super royal 8vo, pp. 1411, with illustrations. Price: $15.00 net.

THE fourth edition of Horsley and Bigger's "Operative Surgery" differs somewhat from the usual textbooks on a similar subject. In 1,387 pages of the two volumes are set forth the views of the authors on what are the most suitable operations for general use.

There is a valuable article on the indications for and method of surgical drainage. Such an epitome of practical information is seldom found in textbooks. It is possible that the space devoted to a discussion on cancer causation might have been more usefully covered in an adequate description of simple operations, such as that for the correction of *hallux valgus*. In this latter connexion too much seems to be taken for granted. The method of treating gangrene of the feet by ligation of the femoral vein could have been omitted or at least more fully commented upon. It is of interest to note that the Russell method of treating fracture of the femur finds much favour with the authors. Homely advice on the problems of compound fracture is presented, and in this and other everyday problems a definite attempt is made to help the beginner in surgery. A large section is devoted to plastic operations. The illustrations in these chapters leave nothing to be desired, but so much space given up to the technique of face lifting and allied procedures is not usual in such textbooks. It is doubtful whether the mutilating and now obsolete operations for radical removal of the tongue are worth so much detail when valuable procedures such as Trotter's pharyngeal resection are omitted. The chapters on chest surgery are well done, and the predilection of one of the authors for heart and lung work finds ample scope.

Australian surgeons would have appreciated at least a note on the surgery of hydatid disease of the lung and liver. There is a very useful chapter on acute conditions in the abdomen and on the modifications of the standard operative methods rendered necessary in cases of emergency. Details of operations for gastroptosis seem superfluous. A good case is made out for not infolding the appendiceal stump with a purse-string suture; the point is rather laboured. Colon surgery is well handled by the senior author, whose experience in this branch of surgery is well known. The method of injection of piles with carbolic acid and almond oil is not mentioned, whereas detailed descriptions of careful suture of the anal region after operation in such cases are given and illustrated. Many surgeons of experience would join issue with these methods. In the surgery of the prostate, Young's perineal operation attracts the authors. No mention is made of the Thomson Walker or of the more recent Harris improvements on the suprapubic operations. Coleman's articles on the surgery of the nervous system are especially commendable. An experienced general surgeon here brings all his resources to bear on the problems of brain and spinal cord diseases. The American advances in this field are nowhere better illustrated than in the reasoned and succinct account of operations for middle meningeal hæmorrhage, cerebral tumours, cranial nerve sections and other procedures which

have proved to be valuable in trained hands. Coleman's reputation as a neurosurgeon is well known, and his contribution to the book is most valuable.

It is regrettable that other sections of the book dealing with subjects of more widespread practical interest are not so complete.

MACROSCOPIC STUDY OF THE BRAIN.

Brain Preparations: A Guide to the Macroscopic Study of the Brain. By J. W. HULTKRANTZ; 1935. London: William Heinemann (Medical Books) Limited. Royal 8vo, pp. 48, with 4 text-figures and 44 figures. Price: 10s. 6d. net.

"BRAIN PREPARATIONS", by Dr. J. W. Hultkrantz, Professor of Anatomy at Uppsala, is a short guide to the macroscopic study of the brain, and has been translated from the first German edition by Professor H. J. Wilkinson, of Brisbane. This small book deals with a special method of dissecting formalin-hardened specimens of the brain, based on blunt dissection by means of glass spatulæ. By this means the author finds that he can demonstrate accurately the extent and course of the white fibres of the brain in a manner which is much more convincing and instructive than by means of the classical method of examination by serial sections. The first chapter deals with the technique employed, and the remaining two chapters with its application to various regions of the brain. The book is profusely illustrated with forty-four photographs of actual dissections which amply demonstrate the value of the method. While there can be little doubt that in expert hands and for museum and teaching specimens the technique would prove excellent, it does presuppose rather more knowledge and care than the average student shows, and it would probably demand a rather abundant supply of material as well as close supervision by trained demonstrators. However, the book is of extreme interest to all those who are interested in the structure of the brain, and should be in the hands of all anatomists and specialists in neurology.

PAGET'S DISEASE OF THE NIPPLE.

Paget's Disease of the Nipple, and its Relation to Surface Cancers and the Precancerous States in General. By K. INGLIS, M.D., Ch.M. London: Oxford University Press. Australia: Angus and Robertson Limited. Imperial 8vo, pp. 233, with 237 illustrations. Price: 54s. net.

THIS book, which is largely a histological study of Paget's disease of the nipple, is a very complete account of the condition from the pathological point of view. In addition, the author has discussed other conditions of the skin which have been confused with, or which have some bearing on the nature of, the condition. Even a cursory examination of the work shows that the writer's experience is very wide, and that he has used the available material to the best possible advantage. Throughout the book there are case histories with the associated histological descriptions of the specimens, which illustrate very clearly the various discussions and arguments.

The first chapter is a very valuable and interesting historical account of the disease and of the hypotheses which have been put forward to explain it. The author then deals with Paget's disease and its relationship to carcinoma of the breast generally. This aspect of the problem is dealt with in various chapters. The differences between the morphological features of Paget's disease and those due to invasion of the nipple region by deep-seated carcinomata are clearly stated. The short chapter on *cancer en cuirasse* will be of value to those interested in breast tumours.

A chapter is devoted to dermatitis and eczema of the nipple and areola. The essential histological differences between these conditions and Paget's disease

are well described and illustrated. There is also a chapter on primary tumours of the nipple which are different from Paget's disease. An interesting account is given of extramammary Paget's disease, and the cases that have been recorded are reviewed critically. Lastly, there are five chapters devoted to pre-cancerous conditions and superficial tumours of the carcinoma type in various parts of the body and also a discussion of certain of the melanomata. The histological feature of the condition, namely, the intraepithelial spread of tumour cells, is discussed throughout the book, and numerous illustrations both of Paget's disease and of other conditions are given.

Professor Inglis's opinion is that the disease is neoplastic from its inception, and that it begins at or near the lactiferous ducts with the epidermis; spread occurs from this region both along the epithelium of the ducts and in the surface epithelium.

Each chapter has a concluding summary of its contents which will be found valuable. The general conclusions are also given in the final chapter.

This excellent book is published on fine art paper, the type is clear, and the large number of illustrations, which are practically all original, are not only exceptionally good, but have been reproduced well. The photographs of specimens or of patients show clearly all the features mentioned in the legends, and the photomicrographs have obviously been chosen and prepared by a keen histologist. The work should be of special interest to pathologists; and because of the general problems discussed it will also be of great value to those dealing with breast and skin diseases—the surgeon and the dermatologist.

PATHOLOGY OF THE THYREOID GLAND.

Hertzler's Monographs on Surgical Pathology: Surgical Pathology of the Thyroid Gland. By A. E. HERTZLER, M.D.; 1936. Philadelphia: J. B. Lippincott Company. Medium 8vo, pp. 316, with 238 illustrations.

THE latest of Hertzler's monographs on surgical pathology, which now number eight, is in characteristic vein. There is no attempt at exhaustive treatment either of the subject or of its literature. The author confines himself, with unusual sharpness, though with a dearth of specific record, to the limits of a personal experience of forty years: this experience is claimed to be sufficiently vast to support some comparatively heterodox opinions on thyreoid pathology and its clinical correlation. The work shows the merits and defects of this individualism. Such experience may warrant some dogmatism, but cannot entirely excuse the somewhat conspicuous neglect and summary dismissal of other work and different lines of inquiry. On the other hand, despite a certain discursiveness, the author's views are forcefully presented and are those of a shrewd and thoughtful observer, who has refused to be trammelled by authority worship and who succeeds in being healthily provocative in statement. This surgeon is also no mean histologist, who believes that "the surgeon's best travelogue is a record of his journeys from the clinic to the laboratory multiplied by the number of days of his natural life", and that "there must be a relation between the pathology and the clinical manifestation"; he dedicates his book to an effort to demonstrate that relation. *O, si sic omnes!*

After a general introductory chapter there is one on general morphology and another on the general pathology of goitre. (In the last of these the author takes liberties with the term fibrin and its derivatives which are, to say the least of it, reprehensible.) In the succeeding chapters he adopts the classification of the American Society for the Study of Goitre into non-toxic diffuse, non-toxic nodular, toxic nodular and toxic diffuse. He begins his study with some observations on goitre in early life, which, though not exhaustively treated, forms a fitting introduction to the general thesis that thyreoid pathology is a process whose development, to be properly understood, must be pursued through all age periods. The author cuts across this already mixed classification with a chapter important

to that thesis on the cardiotoxic goitre, giving timely emphasis and extension to the clinical importance of a phase of thyreoid toxicology that is engaging increasing attention. Incidentally, he joins the school of those who suspect that this phase is not unconnected with degenerative processes in abnormal thyreoids. He describes atypical toxic goitre (interstitial goitre) as a clinical entity probably of polyglandular rather than exclusively thyreoid origin, and correlates it as far as that gland is concerned with colloid changes, interacinar cellular development and lymphoid follicles. He then deals in fairly orthodox fashion with Graves's disease and Basedow's disease (between which he makes distinction) and with thyreoiditis—suppurative, acute and chronic non-suppurative, chronic lymphatic (Hashimoto) and chronic non-specific (Riedel's) types. In the section on myxœdema his challenge of Kocher's *cachexia strumepriva* position should stimulate surgeons to a critical review of their experience of total and subtotal thyreoidectomies and physicians of that with their myxœdema patients. In his chapter on fœtal adenomata he is quite dogmatic, but not correspondingly convincing. The book ends with a chapter on tumours. It is remarkable that a man of the author's experience should never have seen a secondary growth in a thyreoid gland.

The references at the ends of the chapters are given in the form of a commentary on writings that have appealed (or not) to the author. This is an interesting method of supplementing the text, but the author makes no pretentions to completeness of reference. The illustrations are as usual a feature of the monograph and are beautifully reproduced. The legends would in some cases be improved by more specific information. There is a good index. Apart from a few failures in proof emendation, the printing is of the excellence always associated with Lippincott's productions.

MEDICAL HISTORY OF PARRAMATTA.

Medical Practice in Old Parramatta: An Historical Review of Village Doctoring in the Colony of New South Wales. By KEITH MACARTHUR BROWN; 1937. Australia: Angus and Robertson Limited. Double crown 8vo, pp. 152, with illustrations. Price: 12s. 6d. net.

KEITH MACARTHUR BROWN is the fourth in the line of four generations of the family of Brown who have dwelt in "Brislington" and have practised the profession of medicine and surgery in Parramatta. He is therefore well qualified to write the history of medical practice in Parramatta.

In compiling this book he has utilized the records of his family and of the Parramatta District Hospital, as well as the back files of the *Cumberland Argus*; he has also been helped by the staff of the Mitchell Library.

The first medical practitioner to settle in Parramatta was Thomas Arndell, assistant surgeon to the First Fleet which sailed into Botany Bay early in 1788. In the last quarter of 1789, Arndell became surgeon to the newly established settlement at Rose Hill, renamed Parramatta in 1791. Thence onwards Dr. Keith Brown gives a faithful, well authenticated record of the growth and development of the settlement, of the personalities, activities and reactions of its medical men and the various institutions, together with their troubles, trials and defects.

There are descriptions of the conditions existing in the primitive convict hospitals, of the standards of surgery and the disasters attending its practice in those days. The formation of an Australian Medical Association is noted, as is also its decline for want of a medical journal, and the strange reluctance of the members to use the *Australian Medical Journal* then published in Melbourne.

George Hogarth Pringle, M.D., who in 1854 was a fellow resident surgeon of Joseph Lister under Dr. James Syme in Edinburgh, came to Parramatta in 1860, and Dr. Brown asserts that Pringle has a just claim to recognition as a pioneer

in the development of Australian surgery. Pringle imbibed the principles afterwards defined in Lister's monumental papers on 'The Antiseptic System of Surgery", published in *The Lancet* in 1867, and at once introduced them into the routine of his own surgical practice. In 1868 the Royal College of Surgeons of Edinburgh bestowed its fellowship on Pringle. He was a man of charm and culture, loved and admired in a wide circle of patients and personal friends.

In 1878 Dr. Walter Brown first attempted to perform abdominal section for ovariotomy on a woman, aged fifty years, following the procedure laid down by T. Spencer Wells in his book published in 1872. The patient was convalescent in two weeks. The book under review most interestingly records in detail the circumstances leading up to the operation, the operation itself, and the after-care.

Much space is given to the agitation for a new modern hospital, the opposition and ignorance that had to be overcome, the almost endless argument as to details, the battle of sites, until at last a movement, commenced in the late seventies, came to a head in the laying of a foundation stone for a new hospital on June 24, 1896.

Many interesting sketches and anecdotes of the medical men of Parramatta make the book a most attractive one for medical men and others to read. It can hold its place with some English and American books published in the last few years.

The example of Dr. Keith Macarthur Brown should be followed by medical practitioners in other parts of Australia.

WEIGHT REDUCTION.

Weight Reduction Diet and Dishes. By E. E. CLAXTON, M.B., B.S., D.T.M. & H., with recipes by L. BURDEKIN; 1937. London: William Heinemann (Medical Books) Limited. Demy 8vo, pp. 202. Price: 8s. 6d. net.

"WEIGHT REDUCTION DIET AND DISHES" is a book which shows how weight can be controlled, and would serve as a guide to a surgeon who wishes to give some general instructions to his patient as to how he should reduce his weight in preparation for an operation. In this work, general rules for weight reduction, tables of average weights for age and height, food tables, and notes of kitchen recipes are given. The book should be useful.

FOREIGN BODIES AND THE AIR AND FOOD PASSAGES.

Diseases of the Air and Food Passages of Foreign-Body Origin. By C. JACKSON, M.D., Sc.D., F.A.C.S., LL.D., and C. L. JACKSON, A.B., M.D., M.Sc.(Med.), F.A.C.S.; 1936. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical) Proprietary Limited. Crown 4to, pp. 983, with 2,000 illustrations, including 3 plates in colours. Price: 72s. 6d. net.

THIS book is probably unique; we have no knowledge of any similar publication recording such vast experience. A detailed analysis is submitted of over 2,000 foreign bodies in the air and food passages. The whole work is really founded on an even more extensive experience of some 3,000 cases.

This is the third important textbook by Chevalier Jackson, the original being published in 1907, and the second in 1914. It is interesting to observe the advances that have taken place in the science of bronchoscopy and œsophagoscopy during the intervening years, owing largely to the untiring work of Professor Jackson himself and his colleagues.

It occurred to Professor Jackson some time ago to tabulate the details of every foreign body case under the following headings: the patient's age; sex;

nature of the foreign body; location and sojourn; anæsthetic; tube used; problem; forceps used; the point of seizure; the result; the time; the route; and, finally, comment. All this information is now conveniently accessible. It is Jackson's wish that anybody undertaking the removal of a foreign body shall consult these tables before venturing on the operation.

Some interesting facts are revealed on analysing this enormous clinical material. One not entirely devoid of interest is that over 90% of the cases occurred in charity or part-charity patients. Over 80% occurred in patients under the age of fifteen years, and it was possible to attribute the ingestion or inhalation of the foreign body to carelessness in 87% of cases. Of the cases in which foreign bodies were lodged in the lung, 56% were in the right lower lobe. In the œsophagus almost all the foreign bodies were arrested in the cervical œsophagus at the level of the superior aperture of the thorax. In a series of 1,485 cases, the period of sojourn was from one month to forty years. In not a few instances there were multiple foreign bodies in the same patient.

Chevalier Jackson classifies foreign bodies into two great groups: vegetable, and all others. The importance of this is grasped when it is realized that a vegetable foreign body in the air passages kills the patient within a few days, whereas organic foreign bodies, such as bone, may be tolerated for weeks or months without the production of any symptoms. Inorganic foreign bodies, such as those of metal, may cause no symptoms for years. In a series of 200 cases only 70 foreign bodies were radio-opaque, which is a fact of very great practical importance from a point of view of exact diagnosis.

What happens to the lung after removal of a foreign body of prolonged sojourn? This very important question is answered by Professor Jackson, who states that out of 200 cases in which the foreign body was lodged for two months to forty years, all patients recovered perfect health with the exception of four. We quote from the book:

"The recovery of pulmonary tissues from such extensive and prolonged suppurative processes is very remarkable. In some cases it was necessary to dilate the cicatricial stricture to gain access to the foreign-body's removal. On theoretic grounds it seems unlikely that from a single dilatation the lumen would remain dilated: but, of course, it must be remembered that the lumen of the stricture was occupied by the foreign-body which was itself by its bulk a large factor in the obstruction: so that probably the better drainage was due as much, if not more, to the removal of the foreign-body which occluded the strictured lumen.

"Granulations are also an impairment to drainage. Whilst doubtless they do not disappear for some time, yet it is logical to suppose they will gradually recede after the removal of the cause of their presence. Their recession would constantly improve drainage. Repetition of bronchoscopic dilation of a bronchial stricture for the improvement of drainage is very easily carried out without anæsthesia: but, so far, our foreign-body cases have not seemed to require it except in a few instances."

A case is quoted in which a glass collar button had been in the lung for twenty-six years. The patient was emaciated from septic toxæmia and weighed only 98 pounds. Two years after the removal of the collar button she was in perfect health and weighed 182 pounds.

The pathological conditions created by foreign bodies are most elaborately discussed, and also the physical and chemical changes which occur in the foreign bodies themselves. The methods of diagnosis are minutely explained, especially the physical signs and their significance. The technique so successfully used at this clinic is also comprehensively described, and full details are given about the instruments. Much of this information has been published elsewhere.

It is impossible to praise too highly the illustrations and diagrams, most of them drawings made by Dr. Jackson, whose artistic skill is acknowledged. There are also many coloured illustrations from the distinguished author's brush.

The book is indispensable to all specialists in this field, and for many years to come it will continue to remain the most importance source of reference on the subject. We doubt if any work of its size on a medical subject has ever been

published which contains an account of so much original clinical research, observation and experience. The production of the book is in accordance with the very high standard of all Saunders's publications.

SURGERY OF THE SYMPATHETIC NERVOUS SYSTEM.

The Surgery of the Sympathetic Nervous System. By G. E. GASK, C.M.G., D.S.O., F.R.C.S., and J. P. ROSS, M.S., F.R.C.S.; Second Edition; 1937. London: Baillière, Tindall and Cox. Super royal 8vo, pp. 198, with illustrations. Price: 16s. net.

THE appearance of a second edition of Gask and Ross's valuable work on the surgery of the sympathetic nervous system will be welcomed, as it enables the writers to state the opinions they have formed or modified as a result of their own experience and of a survey of an extensive literature in the interval since the first edition was published three years ago. As this branch of surgery is on its trial, a critical study such as is presented by the authors should prove of great help to surgeons who wish to provide the most up-to-date treatment for certain conditions and who desire at the same time to avoid subjecting their patients to useless operations having little or no physiological foundation.

The usual summary of the anatomy and physiology of the sympathetic nervous system concludes by stressing the recent researches of Beattie, Brow and Long and those of Penfield as showing that the sympathetic system is no longer to be regarded as a chain of paravertebral ganglia and outrunning fibres, but as an entity having its headquarters in the hypothalamus or even further forwards in the cerebrum. A salutary note is sounded in the statement that "if we knew what this attribute of temperament signifies . . . we might be considerably nearer an understanding of the fundamental pathology of the sympathetic system". In the material removed in many sympathectomies the authors could find no evidence of any pathological change in the nerve tissue or ganglion cells. Following the methods of Lewis, the writers made careful investigations of cases before and after sympathectomy; they conclude that in Raynaud's disease, structural changes are present in the smaller arterioles and that sympathetic ganglionectomy produces an improvement, but not a complete cure. This applies particularly to cases in which ulceration of the finger tips or phalangeal atrophy has occurred. Pain is relieved, but cyanosis may occur when the fingers are exposed to cold. This has been the experience of some Australian surgeons. It is not to be explained on the grounds of incomplete anatomical removal of sympathetic tissue, but is due to the nature of the changes in the peripheral vessels.

Of six patients with scleroderma and Raynaud's disease only two received benefit from sympathetic ganglionectomy. Cases with hypodermo-lithiasis are examples of gross changes in the peripheral vessels and the benefits from sympathectomy are slight. For *thrombo-angiitis obliterans* the authors advise careful testing before sympathectomy is undertaken. They are not optimistic of its value in this condition. An excellent description of the various types of Raynaud's and obliterative arterial disease is given. "Thorotrast" injection of the femoral artery with X ray examination is regarded as a valuable measure in suspected arterial occlusion.

No mention is made of the useful little operation of dividing peripheral nerves around the ankle for the relief of the severe pain in Buerger's disease. Not all surgeons will agree that amputation above the knee is essential in all cases in which the pulse at the ankle is not palpable.

It is of interest to note that the authors have had successful results in cases of diabetic gangrene, senile gangrene and chronic ulcer by the simple operation of periarterial sympathectomy. They regard this operation as of value and point out that, because its anatomical and physiological basis is not understood, there is no reason to discard a procedure which has proved clinically

successful. Chronic arthritis is classed as one of the diseases which benefit by sympathectomy, and a plea is made for further trial of this operation in both the rheumatoid and osteoarthritic joints. The anterior approach to the stellate ganglion is the one favoured by the authors. Forms of visceral disease relieved by sympathectomy are: Hirschsprung's disease, intractable dysmenorrhœa, cord bladder and renal pain. A variety of operations is described, including presacral neurectomy.

Alcohol injection for *angina pectoris* is discussed, but the writers are not enthusiastic in recommending it. Causalgia is described at length, and a very interesting account is given of cases successfully treated by sympathectomy. This subject is of especial interest to surgeons.

The bibliography at the end of each chapter, the large print, the easy style and the excellent selection of material, all contribute to make the book a valuable addition to the library of surgeon or physician.

A NEW JOURNAL OF SURGERY.

A MONTHLY journal, *Surgery*, devoted to the art and science of surgery, has been published. Its editors are Alton Ochsner and Owen H. Wangensteen. Its editorial board comprises famous American surgeons. The publishers are the C. V. Mosby Company, St. Louis, United States of America. The journal is an outcome of a venture by numbers of young surgeons, and it will fulfil a useful purpose, for it will stimulate the younger men to write. We wish the journal every success.

Proceedings of the Royal Australasian College of Surgeons

QUEENSLAND ANNUAL MEETING.

THE annual meeting arranged by the Queensland State Committee was held in Brisbane on August 11 and 12, 1937. The following programme was presented:

Wednesday, August 11, 1937.

8.15 p.m.—At the Physiology Theatre, University of Queensland. Annual address by Sir John McKelvey: "The Origin of the Gall-Bladder and Some of its Diseases."

Thursday, August 12, 1937.

9.30 a.m.—At the Mater Misericordiæ Public Hospital. Operation by L. M. McKillop: Resection of carcinoma of the pelvic colon.

10.45 a.m. to 11.45 a.m.—Presentation of clinical cases by Fellows of the Mater Misericordiæ Public Hospital:

Milton Geaney: (*a*) Plastic operation to knee. (*b*) Paracaecal abscess following appendicectomy five months previously.

E. D. Ahern: Mass in the left iliac fossa in a girl of nineteen years.

A. S. Roe: Intestinal type of urinary infection.

R. Graham Brown: Laryngectomy.

4 p.m. to 5 p.m.—At the British Medical Association Rooms, Wickham Terrace. Illustrated lecturettes:

1. J. C. Hemsley: "Notes on Goitre."
2. Alan E. Lee: "Primary Carcinoma of the Jejunum."
3. Paul E. Voss: "Fracture of the Forearm, with X Rays." (Specimens sent by Mr. Walker were also presented.)
4. Neville G. Sutton: "Significance of Discharge from the Nipple."
5. R. Graham Brown: "Slides of Cases of Laryngectomy."

SOUTH AUSTRALIAN ANNUAL MEETING.

THE annual meeting arranged by the Committee for the State of South Australia was held in Adelaide on Tuesday, November 2, 1937. A summary of the programme is set out hereunder:

Adelaide Hospital.

3 p.m.—Demonstration of cases.

5 p.m.—Business meeting.

University of Adelaide.

8.30 p.m.—Lecture by Professor F. Goldby: "The Motor Mechanisms of the Fore-Brain." In the Anatomy Lecture Theatre.

TASMANIAN ANNUAL MEETING.

THE annual meeting arranged by the Tasmanian State Committee was held in Hobart from November 5 to 7, 1937. At this meeting the following programme was presented:

November 5, 1937.

12 noon.—Tasmanian Museum. Annual Meeting of Fellows.

3.30 p.m.—Visit to Tasmanian Industrial Gases Proprietary Limited.

8.30 p.m.—Tasmanian Museum. Address: "Safer Surgery in Doubtful Operative Risks", Victor Hurley, of Melbourne.

9.30 p.m.—Address: "Thyroxin Injection Treatment of Otosclerosis", B. Hiller, of Hobart.

November 6, 1937.

9.30 a.m.—Prince of Wales Theatre. Talking films on conservation of vision, J. Bruce Hamilton, of Hobart.

10.30 a.m.—Hobart General Hospital. Clinical meeting, D. W. L. Parker, of Hobart.

3 p.m.—Tasmanian Museum. Address: "Some Aspects of the Surgery of the Colon", C. Gordon Shaw, of Melbourne.

4 p.m.—Address: "The Goitre Problem, with Special Reference to Ætiology and Treatment", Victor Hurley, of Melbourne.

8 p.m.—Hadley's Hotel. Dinner.

November 7, 1937.

10.30 a.m.—Tasmanian Museum. Address: "Injuries of the Knee Joint", C. Gordon Shaw, of Melbourne.

NEW SOUTH WALES ANNUAL MEETING.

THE Acting Honorary Secretary of the New South Wales State Committee reports that the following programme was presented in Sydney on Tuesday, December 7, 1937, at Saint Vincent's Hospital:

General Surgery.

9.30 a.m. to 12 noon.

D. J. Glissan: "Disorders of Growth of Radial Epiphysis", and demonstration of apparatus.

V. M. Coppleson: "Hydatid Growth in the Lung."

W. T. D. Maxwell: Demonstration.

W. Perry: Demonstration.

I. Douglas Miller: "Observations on Arterio-Venous Aneurysm."

V. J. Kinsella: "Observations on Visceral Sensation under Local Anæsthesia: An Unusual Thyreoid Case."

2 p.m. to 3 p.m.

Sir John McKelvey: Operations on the gall-bladder.

3 p.m. to 5 p.m.

V. M. Coppleson: "The Treatment of Acute Cholecystitis."
Sir John McKelvey: Discussion.
I. Douglas Miller: Summary of recent literature on acute cholecystitis.
Dr. Daly: "Anæsthesia in the Bad Risk Gall-Bladder."

Gynæcology.

Operative demonstrations by Dame Constance D'Arcy and F. Brown Craig.

In the evening a dinner was held at the University Club. After dinner, Professor W. J. Dakin, D.Sc., C.M.Z.S., delivered an address entitled: "Biological Studies of the Mechanism of Sex Determination."

Notices.

THE BRITISH POST-GRADUATE MEDICAL SCHOOL.

THE College wishes to draw attention to the announcement of the British Post-Graduate Medical School on page xxx of the advertisements.

NEW DEVELOPMENTS IN SURGICAL EQUIPMENT.

THE attention of Fellows is drawn to pages xviii and xxvi among the advertisements, which illustrate some recent developments in surgical equipment. The Editorial Committee is responsible for the selection of the equipment illustrated thereon. The publishers will be pleased, whenever possible, to supply the names and addresses of the manufacturers to anyone requiring such information.

Editorial Notices.

EDITORIAL communications should be addressed to the Chairman of the Editorial Committee, 57 Collins Street, Melbourne, or to any member of the Editorial Committee. It is understood that original articles forwarded for publication are offered to THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY solely, unless the contrary be stated.

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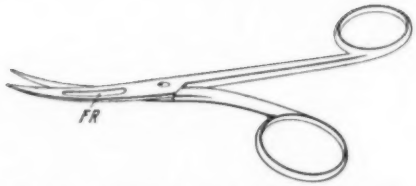

(*Der Chirurg*, August 15, 1937, Number 16, page 640.)

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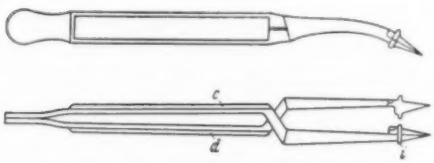

(*Der Chirurg*, August 1, 1937, page 600.)

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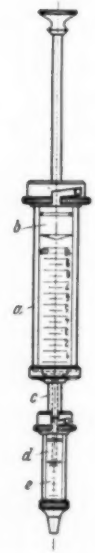

(*Der Chirurg*, August 1, 1937, page 600.)

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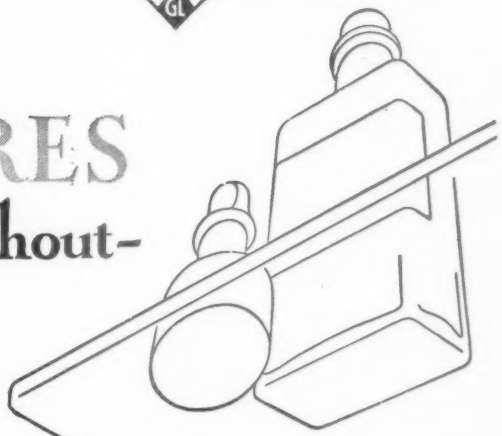

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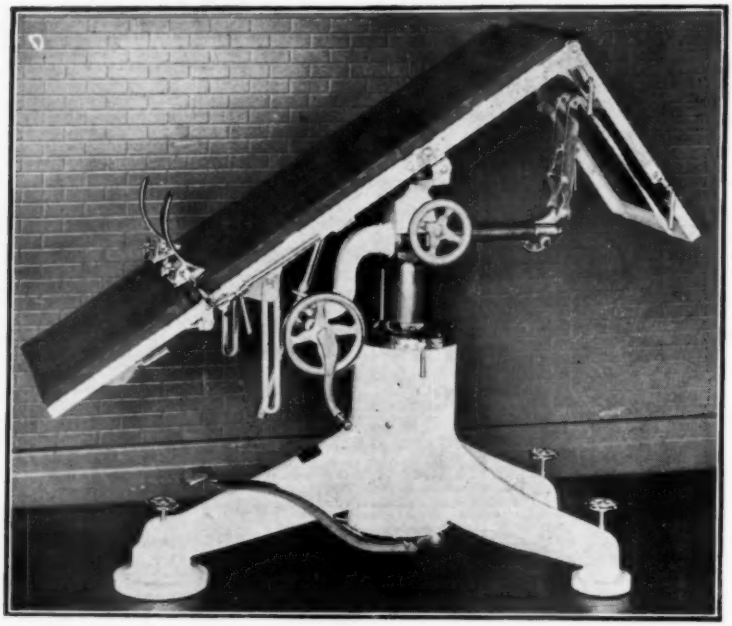

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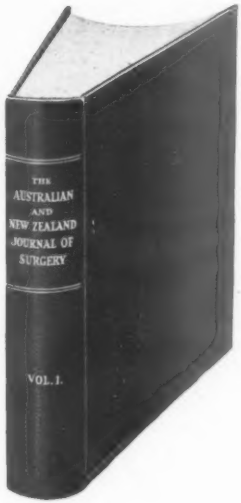

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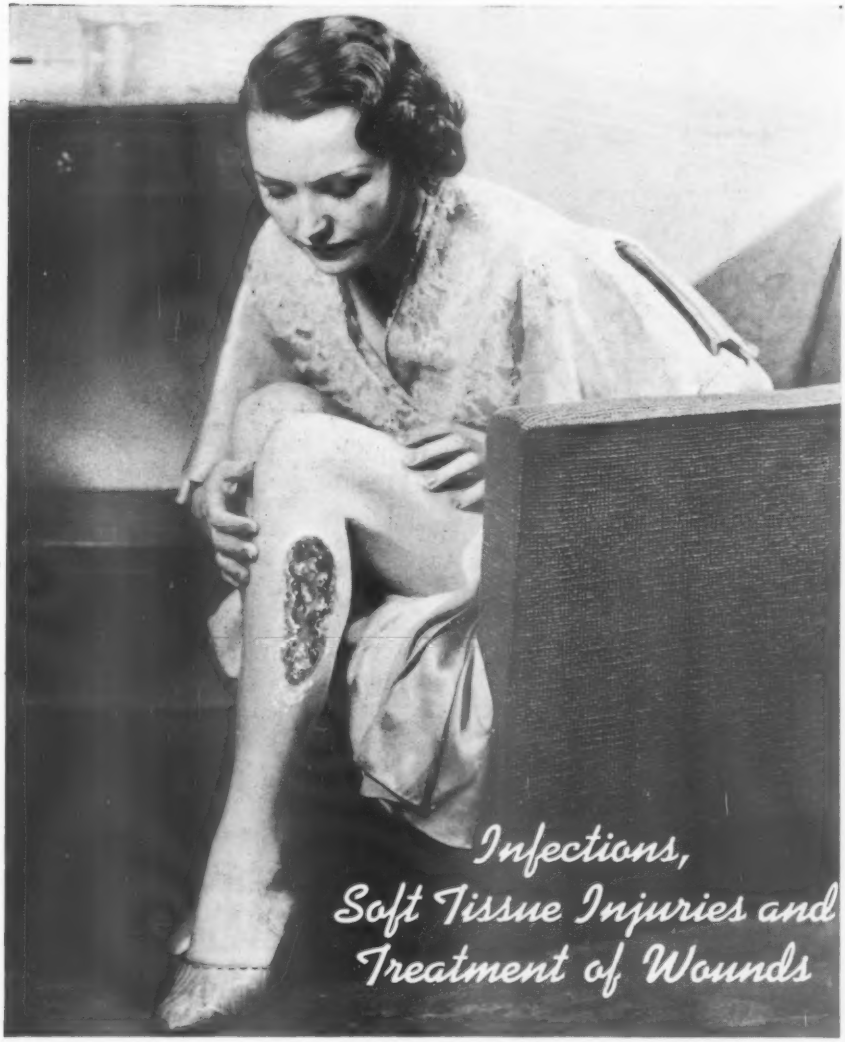

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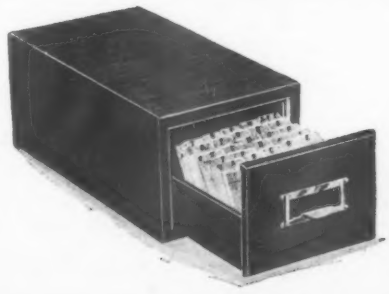

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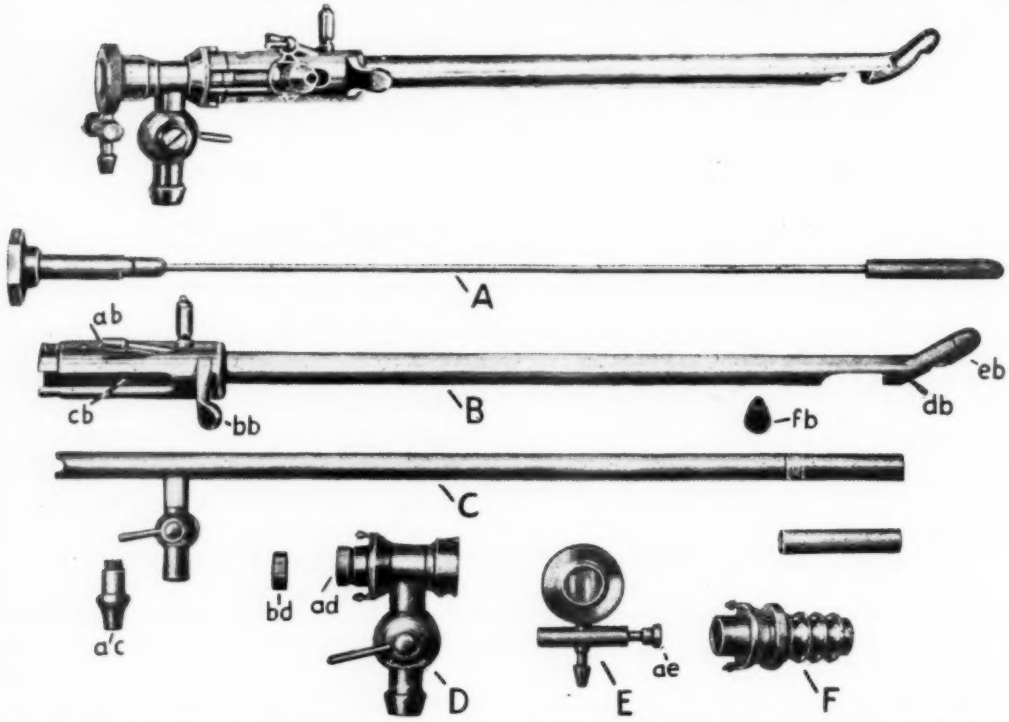

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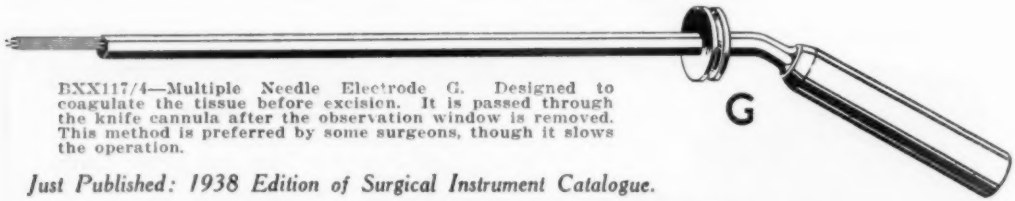

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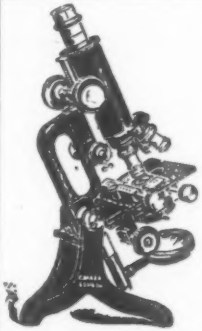

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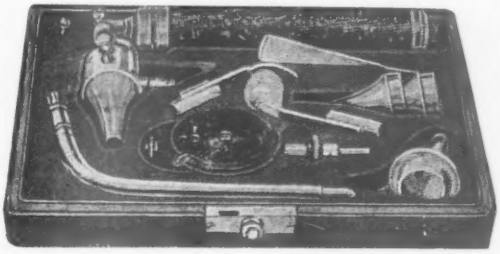

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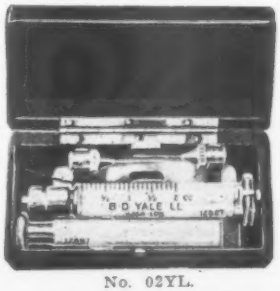

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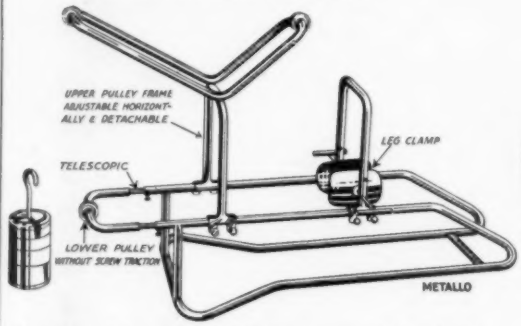

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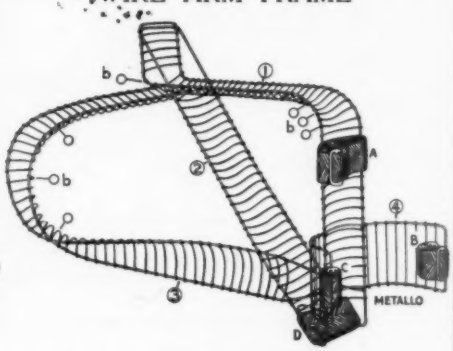

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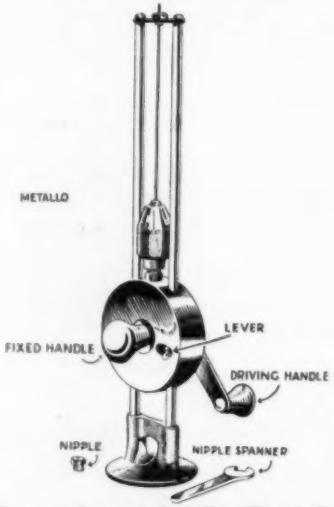

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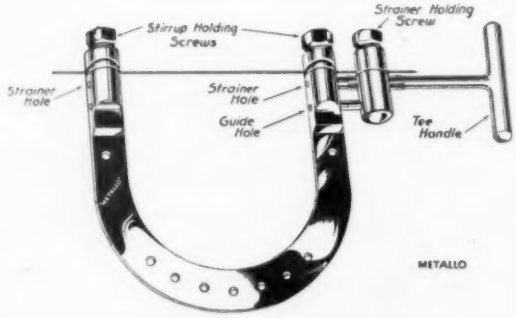

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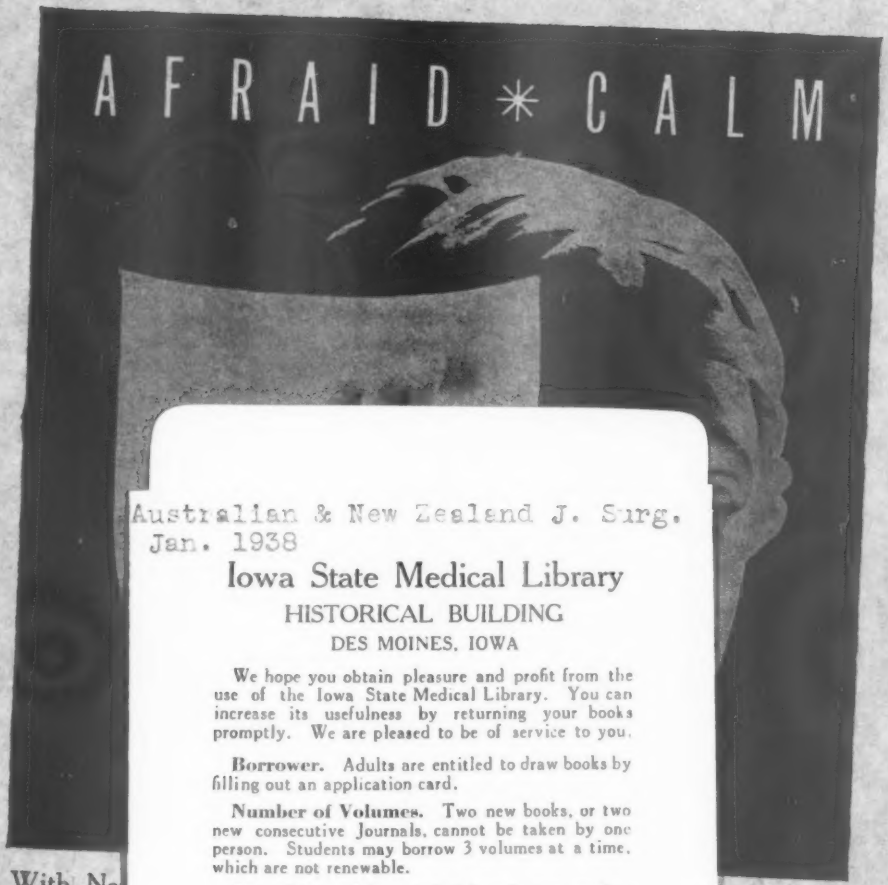

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